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CHICAGO

SUBJECTIVE VISUAL SENSATIONS

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Having been subject to distressing subjective visual sensations during the greater part of my life at intervals varying from three or four days to a month or even longer, I think it is worth while to describe them in detail. I have made numerous notes from time to time and have recorded the effects of various remedies which I have tried in order to obtain relief.

Unfortunately I have had no opportunity of consulting the various books and medical journals, as there is no medical library in Durban, where I reside. However, as I have taken a large number of notes at the time of the attacks, a report may be of some interest and value.

The sensations always take the form of what is commonly called fortification spectra or teichopsia and have never in my case been accompanied with migraine or sick headache. Moreover I have never had any decayed teeth or nasal or pharyngeal obstruction, nor have I suffered from any optical defect or eyestrain. In fact, the only causes to which I can attribute the attacks are anemia and occasional attacks of dizziness, which pass away quickly. My blood pressure is decidedly subnormal; my heart beat and pulse rate are normal and regular, and my arteries are fairly soft.

I must have had several hundred attacks of these visual sensations. They begin without the slightest warning or premonitory symptoms. They never occur when I am in bed but generally take place between meals. Frequently an attack comes on when I am reading. Then I suddenly discover that I am unable to read distinctly with either eye, and I notice a quivering in the air directly in front of me. This usually begins when I have been looking at a white surface or a bright sky. Sometimes it commences when I have been looking through a microscope, whether illuminated by artificial light or by daylight. Then if I look at anything, especially a printed page, I notice that the object or print appears blurred and tremulous. I have frequently made a chart of my fields of vision with the perimeter, but I have never found them to be more contracted than after the symptoms have disappeared. Although the general visual fields are unaffected, the macular fields become exceedingly contracted. Each normal macular field when I am reading print at 10 inches (25 cm.) is an ellipse measuring 25 mm. in the horizontal axis and about 20 mm. in the vertical axis. The area becomes contracted to about 5 mm. in the vertical and 3 mm. in the horizontal axis.

The normal area of critical vision is represented by a large oval. No letter can be clearly seen outside it unless the eyes move slightly. A small oval

represents the contracted area of critical vision during an attack. I have found it better to try the experiment with one eye shut, as otherwise the eyes unconsciously wander away from the fixation letter. Owing to this contraction of the macular field I find it next to impossible to read the words, as I must pick them out one by one, and that only increases the disturbance. Moreover the accommodation becomes greatly weakened, so that only distant objects can be clearly seen, and then only with a feeling of strain.

At the same time the pupils become widely dilated, which confuses my distant vision, as my eyes are myopic (6 diopters) and I continually wear glasses that are slightly undercorrected, as I cannot bear to wear the full correction, a condition very often met with in cases of a high degree of myopia. It is curious that the general field of vision is never contracted either for white or for colors. I may add that the visual field for each eye is abnormally large both for white and for colors, while the vision when corrected is slightly above the normal. I have on many occasions when suffering from an attack had my eyes examined by eminent oculists, but they have never been able to detect any abnormality either in the general appearance of the fundus or in the disk or any alteration in the circulation or caliber of the vessels.

After the contraction of the macular area has lasted for about a minute, or sometimes rather less, I begin to notice a general tremulous motion in everything I see, as though I were looking through a current of hot air or steam. This is immediately followed by the appearance of a zigzag C-shaped figure, the well known fortification spectrum. One spectrum usually appears in front of each eye, and the two are similar in all respects. They are admirably depicted in color in Dr. Liveings' classic work on migraine. My spectra are very similar to those that he has described, except that they do not as a rule show any colors except an intensely bright, almost dazzling gold, which flickers rapidly. The subjective figure is independent of vision; when I close my eyes tightly, I see it much more plainly as a brilliantly illuminated crescent in front of each eye against a black background.

Each of the two spectra forms a C-shaped figure, which as projected into space appears to be about 1 foot (30 cm.) in diameter, apparently much larger than a full moon. The gap in the spectrum seen in front of the left eye faces the nasal side, while that in front of the right eye faces the temporal side. But the figures frequently face the other way, i. e., with both gaps facing left. The zigzag serrations form two rows. Occasionally, three rows are seen, the inner one being exceedingly bright or incandescent. The center one is yellowish, like a gas flame, while the third one, when it is seen, has a slight tinge of red.

The spectra are always in rapid vibration, resembling a field of corn in a strong wind. The motion is always centrifugal and exceedingly rapid. I have made innumerable experiments to try to ascertain the rate of vibration, by comparing the motion with bodies set to vibrate at known velocities. As the result of many trials I have found the rate to be more than 8 and less than 12 vibrations a second.

After about twenty minutes, sometimes less and sometimes longer but never more than half an hour, the figures spread out and become fainter and fainter, until at length they finally vanish. The macular fields increase in area at the same time, until when the images disappear the vision simultaneously returns to normal.

As a rule, two images are seen, one before each eye, and they are identical as regards size, position and shape. It seems to me that there are only two places where such images can originate—at the fovea and at the chiasma, for I have

found that no movement of the eyes can alter the positions of the spectra or cause them to fuse, which naturally must be the case if the stimulus is situated at the chiasma.

Although the disturbance may be traced to the chiasma, it may be that it must be looked for at the fovea itself, or just behind it in the choroid. The fovea and the inner half of the macula contain no trace of blood vessels or even capillaries, the area being nourished by plasma only, since any blood corpuscles would interfere with critical vision. The fact, however, that the two C-shaped crescents are identical in every respect seems to me to point to the chiasma rather than to the maculae,

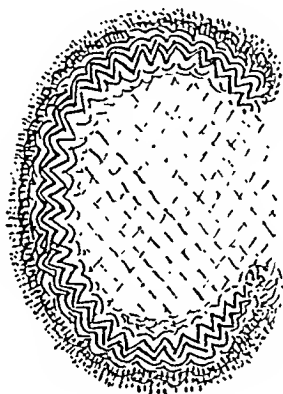


Fig. 1.—General appearance of the fortification spectrum. Drawn from life.

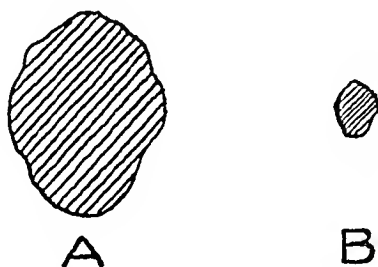


Fig. 2.—*A*, the normal area of critical vision seen at 8 inches (20 cm.). *B*, the contracted area during an attack.

for were the spectra to arise from the two maculae separately, why should they be absolutely the same in all respects? But in either case I think the affection must be traced to anemia dependent in some cases on atonic dyspepsia, affecting the central bundle of nerve fibers, which Prof. Uhthoff traced along the optic nerve to the chiasma. I think it is highly improbable that the origin of the spectra can be traced beyond the chiasma, where the fibers intertwine and partly cross over the optic tracts.

I have experimented with a large number of drugs to obtain relief from the disturbance, but none of them seems to have had much effect. Tincture of cannabis in 5 or 10 minim (0.3 or 0.6 cc.) doses is perhaps

the most effective. Also a cup of strong coffee with a tablespoonful of brandy seems to have a slight effect in hastening the disappearance of the spectra. But by far the most beneficial method that I have found has been to lie down flat and close my eyes or make the room dark. A bandage may be tied over the eyes until the images have vanished (they can be seen even in absolute darkness). If this is done as soon as the symptoms are noticed, the trouble should be all over in about a quarter of an hour.

I think that this disturbance may be attributed either to anemia or to atonic dyspepsia or more often to a combination of the two. When an attack is over I have no symptoms except slight dizziness or faintness, which rapidly disappears.

In conclusion, I wish to draw attention to the importance of plotting the area of critical vision in cases of real or suspected toxic amblyopia arising from any cause.

TRANSITORY WORD BLINDNESS ASSOCIATED WITH RIGHT HOMONYMOUS HEMIANOPIA

REPORT OF A CASE IN A PATIENT WITH CANCER OF THE
PROSTATE GLAND

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DRAMMEN, NORWAY

Dyslexia, or word blindness, is a peculiar psychic disturbance in which the patient is unable to read in spite of normal vision. The defect may be either congenital or acquired. It is present congenitally in about 1 per cent of all persons. It is characterized by a disproportion between the intellectual development of the child and its reading ability, which is affected in varying degree. There may appear a total lack of comprehension of the reading matter. The subject frequently reverses the order of letters or words or may fail to understand certain words. These difficulties may be more pronounced when the child is trying to read fast. Such children are often misunderstood by their teachers, being looked on as obstinate or inattentive.

Acquired dyslexia (alexia, verbal visual agnosia) is also made manifest by the inability of the patient to understand written or printed language, even though the vision is good. As a rule he can understand spoken language without difficulty. The word blindness may be partial, so that the patient may retain, for example, the ability to read written but not printed words, figures but not letters, etc.

The onset of dyslexia may be very sudden. The etiology is diverse, but the pathologic changes are localized in the posterior portion of the outer, upper surface of the angular gyrus of the temporal lobe and the contiguous portions of the occipital lobe. The lesion may often give rise to contralateral homonymous hemianopia. In a pronounced left-handed person, disease localized to the right gyrus angularis, *ceteris paribus*, will give rise to a corresponding condition—left homonymous hemianopia. If the patient states that he is left-handed, yet acquires right homonymous hemianopia, it is reasonable to believe that he is not entirely left-handed by nature.

The case to be reported is of interest not only from the ophthalmologic, but from the psychologic, and even the philologic, point of view.

REPORT OF CASE

A man, aged 65, apparently healthy and strong, came to see me for the first time on May 15, 1935. His complaint dated from the previous April 17, at which time he first noticed a disturbance in his ability to read. On reading a letter

which he had dictated to his stenographer he was astonished to find that it contained numerous invectives. When read aloud to him by the stenographer, however, it sounded precisely as he had dictated it. The patient, immediately aware that something was wrong with his mental processes, tried to find certain well known names in the telephone directory. Some of these he could find and others he could not. His general health had always been good except that for the past

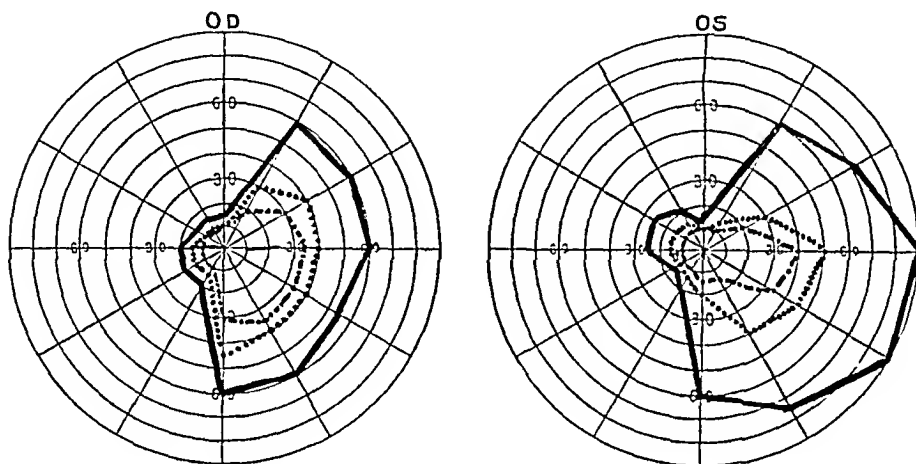


Chart 1.—Visual fields, showing right homonymous hemianopia. The solid line indicates the results of the examination with a 2 mm. white test object; the dotted line, the results with a 2 mm. red test object, and the dash and dot line, the results with a 2 mm. green test object.

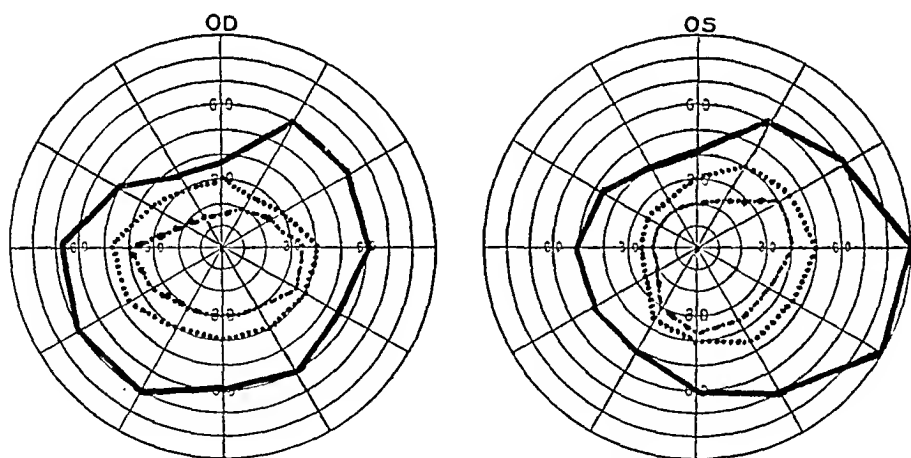


Chart 2.—Visual fields taken five weeks after those in chart 1, showing considerable improvement. The solid line indicates the results of the examination with a 2 mm. white test object; the dotted line, the results with a 10 mm. red test object, and the dot and dash line, the results with a 10 mm. green test object.

year he had had symptoms referable to prostatic hypertrophy. For these, he was now undergoing a series of roentgen treatments. He was left-handed.

Examination of the eyes revealed normal findings. The vision in the right eye with a $+1.50$ sphere was 7/10, and that of the left eye with a $+2$ sphere, 7/7. Examination of the visual fields showed a pronounced right homonymous hemianopia, especially for colors, more marked in the left eye (chart 1).

On reading the patient made many characteristic mistakes. He consistently read b for p, g for k, t for d and f for p. Some words he could not read at all, yet he understood them when they were read to him. There was no aphasia.

The prognosis was considered good, and potassium iodide was prescribed. Five weeks later the patient returned, obviously a doomed man. He was cachectic and had a bad color. The ocular changes remained the same except that the visual fields showed considerable improvement (chart 2). He was now able to read any text faultlessly. The blood pressure was found to be about 180 systolic and 100 diastolic. Examination of the urine did not reveal any albumin or sugar.

Two months later the patient died from prostatic cancer. As autopsy was not performed, it is impossible to state definitely whether the hemianopia and dyslexia were due to a carcinomatous metastasis or to cerebral vascular disease. The increase in blood pressure might point to the latter possibility.

In the literature to which I have had access I have found no case entirely similar to this one. To me, the interesting feature is the peculiar way in which the dyslexia manifested itself. It seems fair to assume that the patient had a certain amount of ill feeling toward the person to whom the dictated letter was addressed. Under ordinary circumstances this ill feeling was suppressed and hidden beneath conventional polite phraseology. The patient's acute cerebral disturbance, however, interfered with his appreciation of certain word pictures, whereupon he promptly substituted the word images of the invectives he had always been tempted to use:

Such a condition might give rise to legal problems, for instance: The patient writes a letter himself, by hand. He does not know anything about his own ailment, this having arisen shortly before. In his letter he writes invectives instead of polite phrases. Is he then juridically responsible for his blunder? Or the patient writes a letter to B. In his letter he mentions A, whom he abuses. In good faith B uses the expressions about A. Who, then, is the responsible party—the patient or B? The examples might be multiplied indefinitely.

Another peculiarity of the case reported here is that the patient in interchanging consonants strictly followed the so-called phonetic law of Verner. As will be recalled, Jacob Grimm, a German, enounced his phonetic law in 1822. In 1876 this was amplified by the Dane Karl Adolf Verner in his famous work "*Die Konsonantverschiebungen der indogermanischen Sprachen.*" Starting with Sanscrit, Verner proved that there was a constant and regular consonant alteration traceable through the Greek, Latin, old Norwegian, German and Scandinavian languages. Thus *bhiter* in Sanscrit became *πατήρ* in Greek, *pater* in Latin, *vater* in German, father in English and *fader* in the Scandinavian languages.

Finally I want to mention that the considerable improvement in the patient's fields of vision may have been due to the fact that he was partially left-handed. As the homonymous hemianopia was right-sided he could hardly have been absolutely left-handed.

RELATION BETWEEN BLUE SCLERAS AND HYPERPARATHYROIDISM

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AND

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The clinical entity consisting of blue scleras, osteoporosis with spontaneous fractures and deafness and the dominant hereditary transmission of the syndrome have long been known to ophthalmologists and pediatricians. The association of blue scleras with fragilitas ossium was noted by Ekman (1788), Lobstein (1833) and von Ammon. Van der Hoeve and de Kleyn¹ and Bronson² independently included hereditary deafness (which, according to some observers, may develop only after the age of 20) as a part of the entity. German authors commonly refer to this symptom complex as the van der Hoeve and de Kleyn syndrome, while in the English literature there is a tendency to link the entity with the name of Lobstein.

It is not pertinent to the purpose of this article to delve into the early historical facts or to decide to which authorities credit should be given for advancement in the knowledge of the syndrome of blue scleras. An excellent and exhaustive historical review was recently made by Dessoiff.³ In our search of the literature confined to data bearing on the etiology we found varied and conflicting theories, in which lies a potential danger too often overlooked. Conclusions loosely drawn or founded on insufficient facts may lead to treatment, even operative intervention, involving serious consequences.

In the Netherlands and England, where cases of the syndrome have been frequently reported, the explanation was generally made that the condition is due to a deficient development of the mesenchyma, since most of the organs affected are of mesenchymal origin (Eddowes, Peters, Freytag). In recent years many authors have considered that alteration in the ductless glands may be the causative factor (Behr, Bolten, Gutzeit, Hofmann); others, like Takahashi⁴ and Kaznelson,⁵

1. van der Hoeve, J., and de Kleyn, A.: *Arch. f. Ophth.* **95**:80, 1918.

2. Bronson, E.: *Edinburgh M. J.* **18**:240, 1917.

3. Dessoiff, J.: *Blue Sclerotics, Fragile Bones and Deafness*, *Arch. Ophth.* **12**:60 (July) 1934.

4. Takahashi, T.: *Arch. f. Ophth.* **115**:206, 1925.

5. Kaznelson: *Zentralbl. f. d. ges. Ophth.* **18**:710, 1918.

made an attempt to explain the syndrome as entirely caused by hypo-functioning of the parathyroid glands, involving hypocalcemia. In contradiction of this view, Dessoff pointed out the possibility that hyperparathyroidism might be the sole cause. The comparison and evaluation of known facts, in addition to further investigation, are essential to an understanding of this uncommon symptom complex. We have made a detailed study of 2 cases that came under our observation, with the purpose of throwing light on the relationship between the syndrome of blue scleras and that of hyperparathyroidism.

THE SYNDROME OF BLUE SCLERAS

A marked hereditary tendency is inherent in *fragilitas ossium*, a condition characterized by imperfectly generalized ossification of all the bones. In these cases the child may be born dead, showing multiple fractures acquired in utero, or these may not develop until soon after birth. The number of fractures is at times outstanding. A child 15 months old who came under our observation had eight fractures, according to a single roentgenogram of the lower extremities. In other instances children who are apparently normal at birth do not show evidence of brittleness of the bones in early infancy, but this develops after a delay, in later childhood or early adolescence. There is a tendency for the condition to become arrested after puberty and gradually recede or disappear as time passes. Boyd⁶ mentioned, in addition to the foregoing characteristics, osseous swellings that sometimes occur in the occipital and frontal regions and especially in the temporal region, so that the ears are turned outward and down. Furthermore, owing to incomplete ossification, the skull appears as a mere membranous bag or a few osseous plates. If ossification proceeds somewhat further the skull may present a large number of wormian bones.

In exceptional cases the hereditary tendency may be absent, as in one case reported by Gutzeit,⁷ but a study of the antecedents invariably shows the anomaly through many generations. Investigation into the family tree discloses the occurrence of the inherited syndrome throughout as many as four or five generations, such instances being fairly common (Adair-Dighton, Berneaud, Bronson, Burrows, Cockayne, Conlon, Knapp, Paul, Peters, Spurway, Stephenson-Harman, Stobie⁸). In some of the affected subjects all three symptoms of the syndrome were present; in others, only one or two. Particularly in the older literature,

6. Boyd, W.: *A Text-Book of Pathology*, ed. 2, Philadelphia, Lea & Febiger, 1934.

7. Gutzeit, R.: *Klin. Monatsbl. f. Augenh.* 68:771, 1922.

8. Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1932.

the presence of otosclerosis is often not reported. Only in comparatively recent years was attention called to the combination of deafness with fragilitas ossium and blue scleras. Furthermore, the deafness, which in these cases is hereditary, develops often after the age of 20 and is due to secondary changes suffered as sequelae of the multiple fractures, or, owing to a possibly lessened general resistance, life is usually terminated by an intercurrent disease, such as pneumonia, before the deafness manifests itself. The close association of the two cardinal symptoms, i. e., idiopathic spontaneous fractures and blue scleras, was recognized definitely by Eddowes.

The blue color of the scleras resembles that met with in staphyloma sclerae. The intensity of the hue varies with, and is more or less accentuated in, individual cases. As a result different authors have used varying descriptive terms, such as blue-gray, sky blue, blue with a yellowish tinge, china blue, brilliant blue and intensive blue. The discoloration is usually diffuse, beginning at the limbus and continuing backward. In one of Hirschmann's cases⁹ the patient had a ring of normal color about 2 or 3 mm. wide around the limbus; then it shaded into intensive blue. Among our patients one showed a marked deep blue ring-shaped area surrounding the limbus, which gradually changed into a less intensive blue. In cases of intensive blue scleras the contrast to the normal coloring is extremely marked. In cases of mild involvement, however, differentiation from the normal may be difficult, since many new-born children and even adults, especially those of dark-pigmented races, occasionally show a slight but definite bluish color of the sclera. Abnormal translucency (Gutzeit, Blatt, Freytag) is not an absolute criterion. There are borderline instances in which variation of a pathologic blue color from the normal is so insignificant as to cause some hesitation in making a diagnosis of blue scleras.

The underlying pathologic condition is either abnormal thinness of the sclerotica or lack of deposits of calcium within the outer coat of the eye. Buchanan reported that in this condition he noted that the thickness of the cornea was three fourths of the normal, and that of the sclera, one third of the normal, with absence of Bowman's membrane and diminution of the fibers, observations which were recently confirmed by Casanovas.¹⁰ These observations were contradicted by the results of anatomic examination in a case of blue scleras reported by Bronson and by the observations made by Wirth and Vogt with the aid of the slit lamp. It therefore can be readily assumed that the blue coloration must be only the result of a changed transparency to light, no anatomic change being microscopically discernible.

9. Hirschmann, J.: *Ztschr. f. klin. Med.* **126**:718, 1934.

10. Casanovas: *Zentralbl. f. d. ges. Ophth.* **31**:580, 1934.

With blue scleras and spontaneous fractures there are frequently further complications of the bones, such as prominent frontal and occipital bones (Boyd, Blatt, Bronson, Stroble), frequent luxations of the interphalangeal joints, especially of the thumb (Behr, Cockayne, Conlon, Crocco, van der Hoeve), and kyphosis, lordosis and scoliosis. The deafness may be attributed to otosclerosis due to changes in the petrous bone and calcification in the labyrinth (Stenvers, Stobie), combined with partial stapes ankylosis (Bigler, Gimplinger). Disturbance of the calcium metabolism, resulting in late dentition (Crocco, Paal), and brittleness of the teeth (Paal, Stobie, Terrien) may also be associated with the condition. Other complications may be present, such as syndactylism (van der Hoeve), mongoloid idiocy (Bolton), vitium cordis, palatum fissum (Voorhoeve), conical cornea (Behr, Urbanek) and zonular cataract (Blegvad and Haxthausen)

HYPERPARATHYROIDISM

In order to place the syndrome of blue scleras within or outside the domain of endocrine disturbance, especially hyperparathyroidism, it is necessary to review the known facts concerning parathyroid disorders. This knowledge has been extensively modified in the last decade. As in the development of many other medical problems, orientation is rather difficult because of contradictory observations and unjustified conclusions. It would lead far afield to give all the details concerning parathyroid disorders. Therefore, only those essential to the understanding of the subject are here discussed.

The pioneer work in this field was done by MacCallum and Voegtlin, who discovered that in parathyroidectomized dogs the calcium content of the blood serum is reduced. This was later strongly confirmed by further research. Since the isolation of the active principle of the parathyroid gland by Hanson (1924) and Collip (1925) it has been repeatedly demonstrated that the injection of parathyroid extract produces marked elevation of the blood calcium level. Brehme and György found, in addition, that parathyroid extract lowers the blood phosphate content and causes a shift in the p_H of the blood toward the acid side without disturbing the carbon dioxide-combining power of the blood. Reiss and Aub, working independently, found lowering of the blood phosphorus level due to injections of parathyroid extract. The various authors unanimously agreed that such injections in normal persons result in a rise of the blood calcium content above the physiologic level, accompanied by an increased calcium content of the urine (even to six times the normal). The increase in calcium salts they considered to be due to their mobilization from the bones, which store compounds of this element just as the liver stores glycogen. Thus the conclusion

is obvious that the physiologic function of the parathyroid glands is the exact regulation of the calcium metabolism, i. e., control of the rate at which the calcium is moved from its deposits, namely, the bones, into the blood stream and tissues and, finally, into the urine. The amount of calcium present in the tissues is responsible for their neuromuscular irritability, which varies inversely with the calcium content (hypotonia of the muscles is associated with hyperparathyroidism and hypertonia of the muscles with tetany). Pathologic disturbance of the parathyroid glands may lead to either hyperparathyroidism or hypoparathyroidism, the former resulting in the presence of a negative calcium balance and generalized osteitis fibrosa cystica, and the latter, in tetany accompanied by a positive calcium balance (Goerner and Samuelsen¹¹) with characteristic hyperirritability, known clinically as the Chvostek and Trousseau signs.

The increased activity of the parathyroid glands, as has already been mentioned, results in hypercalcemia of the blood arising from excessive loss of calcium from the body by way of the kidneys, the level of calcium in the blood, however, being only a rough indication. The exact evaluation of the calcium metabolism can be obtained solely by careful study of the calcium balance. Hunter and Turnbull¹² strongly advocated determination of the calcium balance and stressed the comparison that "just as the level of a river does not indicate the direction in which it flows, so the levels of the calcium in the blood give us, in themselves, no indication as to whether the calcium is flowing into the excretory channels or into the tissues."

Method of Estimating the Calcium Balance.—In order to establish the calcium balance the patient has to be kept on a fixed diet of sufficient caloric value with a low calcium content (100 mg. a day). The intake of calcium must be closely observed, and only distilled water should be used both for drinking purposes and in the preparation of meals. In addition, the intake of sodium chloride has to be controlled. Sufficient sodium bicarbonate should be given by mouth to make the reaction of the urine neutral, since with an acid medium the figures for calcium are necessarily higher. As the calcium is usually eliminated through the kidneys, the calcium content of the urine has more significance than that of the feces. Only in pathologic conditions, such as calcification of the kidneys (which may be associated with deposits of calcium in other organs, such as the lungs and spleen, in hyperparathyroidism), is there no increased excretion of calcium in the urine, for, owing to the inability of the kidneys to eliminate the extra load, it will appear in the

11. Goerner, A., and Samuelsen, G.: Calcium Metabolism in Idiopathic Hypoparathyroidism, J. A. M. A. **102**:1001 (March 31) 1934.

12. Hunter, D., and Turnbull, H. M.: Brit. J. Surg. **19**:203, 1931.

feces. In such cases the quantity of calcium in the feces must be determined. The most reliable method seems to be either that of Kramer and Tisdall or the modification of this by Clark and Collip.

The normal content of calcium in the blood is between 9 and 11 mg. per hundred cubic centimeters. Increase of the blood calcium level with simultaneous increase of the excretion of calcium, when the intake is controlled, is called a negative calcium balance and is characteristic of hyperparathyroidism. The injection of an extract containing the active principle of the parathyroid gland in normal persons and in animals produces a similarly high calcium level in the blood serum and an increase of excretion of calcium in the urine, not infrequently reaching a figure six times the normal level.

Rôle of the Phosphorus Content.—In hyperparathyroidism the high level of the calcium in the blood and the increased excretion of calcium in the urine are associated with a low level of phosphorus in the serum. This glandular hyperactivity is almost unique in producing these simultaneous changes. Numerous conditions may cause an increase in the calcium level of the blood (multiple myeloma and metastatic malignant growth) or a low phosphorus content (rickets and osteomalacia), but the singular combination of an increased calcium content and a low phosphorus content is pathognomonic of hyperactivity of the parathyroid glands. According to Aub and Bauer, a serum calcium level above 11 mg. per hundred cubic centimeters and a phosphorus level below 3.5 mg. per hundred cubic centimeters should be regarded with suspicion, especially if repeatedly noted.

Method of Determining the Phosphorus Content.—Fresh blood should be used in making the determination of the phosphorus content, for if the blood has been allowed to stand the phosphorus content shows variation. When the corpuscles have been laked the liberated organic esters are rapidly hydrolyzed by the plasma phosphatase, and the concentration of the inorganic phosphates rises rapidly. Therefore, the blood should be immediately oxalated, and hemolysis should not be permitted. With such precautions the experimental error in the method of Briggs (used by Hunter and Turnbull) should not exceed 2 per cent, a phosphorus content of between 2.5 and 3.5 mg. per hundred cubic centimeters being considered normal. Aub and Bauer used the method of Frike and Subbarow and regarded any level below 3.5 mg. as indicative of hypophosphaturia.

In seventy-three of seventy-eight cases reported by various workers, all of which were unquestionable instances of hyperparathyroidism, determinations consistently showed a serum calcium content above 11.5 mg., and in 59 cases there was a constant level above 12 mg.

Phosphatase.—The hypercalcemia and the associated decrease in the inorganic phosphorus content are accompanied by increased activity of the blood phosphatase. The latter is an enzyme that plays a specific rôle in the deposition and maintenance of the calcium and phosphorus compounds in the tissues. An increased concentration of the enzyme in the plasma is found in several diseases in which there is abnormal destruction or formation of bone tissue, such as Paget's disease, mild and healing rickets, certain types of osteomalacia and obstructive jaundice. Very high values for phosphatase are encountered particularly in active rickets and generalized Paget's disease. However, only hyperparathyroidism is known as a clinical entity in which a markedly increased phosphatase content in the blood serum (from 12 to 25 Bodansky units) is combined with a negative calcium and a negative phosphorus balance. The phosphatase level of from 2 to 4 units is considered normal and is probably an index of the osteoblastic activity. It is elevated in hyperparathyroidism in direct proportion to the extent of the osseous disorder and is independent of the degree of parathyroid disturbance. Accordingly, osseous disturbances of various origins may result in increase of the phosphatase content, but in such cases there will be no negative calcium and phosphorus balances. Observation of persons after operation for parathyroid tumors shows a dramatic change in the calcium and the phosphorus metabolism, but, on the other hand, the phosphatase level, after a period of months, only gradually returns to the normal.

Interpretation of Findings.—The variation between what is pathologic and what is physiologic, especially at the beginning of any disease, may be so slight that differentiation is not sharp. Hyperactivity of the parathyroid glands may be present without an appreciable increase in phosphatase activity in cases in which the alterations in the osseous substance are not yet far advanced or in those instances in which shifting of the calcium and phosphorus levels can be detected only after repeated examination. However, the laboratory findings of an increased blood calcium level and a decreased blood phosphorus level, associated with increased excretion of calcium and phosphorus in the urine and an elevated phosphatase level of the blood, should strongly suggest hyperactivity of the parathyroid glands.

Osseous Condition.—It may be definitely stated that the characteristic laboratory findings demonstrate a profound disturbance in the calcium metabolism, viz., the inability of the bones to store and retain calcium, which is the most outstanding symptom of parathyroid hyperactivity and must result in generalized decalcification of the skeleton. The typical skeletal changes were first described by von Recklinghausen¹³ as osteitis fibrosa cystica, and thirteen years later Askanazy

13. von Recklinghausen, F. D., in *Festschrift für Rudolf Virchow*, Berlin, G. Reimer, 1891.

first pointed out the relationship of this condition of the bones to a tumor of the parathyroid gland in his case. Since then further observation, study and isolation of the parathyroid hormone have supplemented the knowledge of the physiology of the parathyroids and proved that Recklinghausen's disease is a clinical manifestation of parathyroid hyperactivity and that an identical osseous disorder can be produced experimentally in animals by injections of parathyroid hormone.

Generalized osteitis fibrosa consists of widespread resorption affecting all the bones, resulting in osteoporosis, cyst formation and benign giant cell tumors the cells of which are similar to the osteoclasts normally present in Howship's lacunae. The excessive stimulation of osteoclastic resorption is accompanied by new formation of fibrous tissue occupying the dilated haversian canals, i. e., marked fibrosis of the marrow (in the recent German literature osteitis fibrosa cystica is referred to as osteodystrophia fibrosa). Some of the connective tissue may become converted into osteoid tissue, formation of new bone taking place with rows of osteoblasts, but resorption greatly preponderates and outstrips ossification and apposition, while osteoclasts and giant cell tumors grow to larger proportions. The presence of osteoclastoma or giant cell tumors is a characteristic feature of the picture of hyperparathyroidism. The newly formed connective tissue is poorly vascularized, and as a result degeneration, softening and cyst formation frequently occur. The osteoclastomas and cyst formation are important factors in differentiating osteitis fibrosa cystica from generalized osteoporosis, and one must bear this in mind in the discussion of the latter disease.

A negative calcium balance and generalized osteitis fibrosa cystica are the distinguishing features of parathyroid hyperactivity, which may be caused by a malignant growth or by so-called adenoma of these glands. Other clinical features may be found, among which Bauer¹⁴ enumerated polydipsia, polyuria, general malaise, constipation, anorexia, loss of weight, vague muscular and articular pains, tenderness of the bones, frequent fractures, decreased excitability of the nerves, skeletal shortening, kyphosis, osseous tumors, stones in the kidney and ureters and, frequently, anemia with leukopenia. The osseous changes consist of generalized decalcification, cyst formation and tumors, as was previously mentioned.

The disease entity which was primarily known as Recklinghausen's disease and later known as osteitis fibrosa cystica and hyperparathyroidism was first recognized by Mandell in Vienna and by DuBois in America. At the present time the literature contains reports of more than a hundred cases in which the clinical and the laboratory diagnosis

14. Bauer, W. J.: J. Bone & Joint Surg. **15**:135, 1933.

have been confirmed by operation. The pathologic observations and ensuing postoperative recovery jointly clarified the picture. In the hundred cases in which an operation was performed, about one fifth of the patients were under observation and operated on at the Massachusetts General Hospital. The work of Churchill, Albright and Bauer and their co-workers,¹⁵ therefore, should have much weight in a discussion of the subject. Churchill emphatically warned against the danger of overstepping in this new field of endocrine disturbance and advocated as next in importance to the study of various clinical symptoms the careful consideration of the calcium metabolism and the roentgenographic observations on the bones.

This author also took an emphatic stand against the removal of one or more of the parathyroid glands (including interference with their blood supply) for several conditions other than hyperparathyroidism and called the evidence supporting such recommendations highly inconclusive. He referred particularly to the work of Oppel¹⁶ and of Ssamarin, who performed parathyroidectomy in about seventy cases of ankylosing polyarthritis. Leriche, and Ballin, in this country, resorted to the same treatment. The latter was even more radical, advocating parathyroidectomy for Paget's disease as well as for ankylosing polyarthritis. This is of great importance in consideration of the types of cases in which there are blue scleras and osteochondritic changes. The latter condition is usually regarded as a form of arrested embryologic development and therefore is excluded from the endocrine type of disorders. Ballin was carried away by the conception that hyperparathyroidism, either fetal or acquired early in life, may be the responsible factor in the mechanism involved in these cases and advised reinvestigation of all juvenile types of osseous disturbances, particularly fragilitas ossium and achondroplasia.

DIAGNOSIS OF PARATHYROID HYPERACTIVITY

There is a wide difference between the opinions of Churchill and Ballin. The former insisted that the classic picture must be present before parathyroidectomy is indicated; the latter drew criteria for surgical intervention much wider in scope. A pertinent question naturally

15. (a) Albright, F.; Aub, J. C., and Bauer, W.: Hyperparathyroidism: A Common and Polymorphic Condition as Illustrated by Seventeen Proved Cases from One Clinic, *J. A. M. A.* **102**:1276 (April 21) 1934. (b) Churchill, E. D., and Cope, O.: *Surg., Gynec. & Obst.* **58**:255, 1934. (c) Albright, F.; Bloomberg, E.; Castleman, B., and Churchill, E. D.: Hyperparathyroidism Due to Diffuse Hyperplasia of All Parathyroid Glands Rather Than Adenoma of One, *Arch. Int. Med.* **54**:315 (Sept.) 1934.

16. Oppel, W. A.: *Endokrinologie* **6**:11, 1930.

arises, viz.: What factors led to such opposing views of the indications for operative intervention on the parathyroid glands?

Churchill and his co-workers based their diagnosis of parathyroid disturbances mainly on the evidence of a negative calcium balance combined with the characteristic roentgenographic findings of the skeleton. In their evaluation of symptoms the associated clinical manifestations, which were enumerated by Bauer, were considered of secondary importance. Certain other clinical features may be either present or absent during the course of the disease in different cases. These are calculus formation in the kidneys, muscular atony and changes in the terminal joints of the fingers (which, according to Keynes and Taylor, are very short and square, with correspondingly stubby and broad nails). The slightly bulbous finger ends should not be confused with the clubbing seen in pulmonary osteo-arthritis. The most reliable diagnostic evidence of osteitis fibrosa cystica is obtained by roentgenography.

Decalcification of the bones may occur in various diseases and has been observed in association with disuse, arthritis, fractures, starvation, rickets, osteomalacia and celiac disease associated with rickets (all of which are apparently dependent on vitamin D deficiency), but these forms of decalcification are entirely different from that in osteitis fibrosa cystica and do not belong in the same group. Camp¹⁷ insisted that osteoporosis alone is not sufficient evidence on which to base a roentgenographic diagnosis of parathyroid disorder and described the osteoporosis in osteitis fibrosa cystica as uniform and granular, stressing the fact that its peculiar form of mottled atrophy is distinct from the ordinary type seen in osteoporosis associated with acute and chronic disease of the bones and neurotrophic conditions.

In contradiction to definite laboratory findings, Oppel, Leriche, and Ballin¹⁸ failed to establish an accurate calcium balance and were satisfied with an occasional determination of the calcium content of the blood serum. Ballin, following Oppel, emphasized the importance of muscular hypotonia associated with parathyroid disturbances and pointed out four distinct diagnostic signs: (1) the determination of the number of milliamperes necessary to provoke muscular contraction; (2) chronaxia, which was introduced by Lepique, as an electric time coefficient of neuromuscular excitability (according to Bourguignon, increase of chronaxia in hyperthyroidism is more reliable than the serum calcium content); (3) demonstration in a motion picture of the slow or weak action of muscles, and, finally, (4) shortening of the RT wave in the electrocardiogram, due to myocardial weakness. Ballin attached great signifi-

17. Camp, J. D.: *Osseous Changes in Hyperparathyroidism*, J. A. M. A. **99**:1913 (Dec. 3) 1932.

18. Ballin, M.: *J. Bone & Joint Surg.* **15**:120, 1933; *Ann. Surg.* **96**:649, 1932; **98**:868, 1933.

cance to muscular hypotonia in establishing the diagnosis and included in the syndrome of hyperparathyroidism clinical entities varying from typical generalized osteitis fibrosa cystica, Paget's disease in his opinion merging into it without any striking differences. Such an assertion does not seem to be valid, considering all the known evidence that the two diseases are distinct entities, and stands in contradiction of the findings of Schmorl, who in a careful investigation of one hundred and ninety cases of Paget's disease failed to encounter symptoms of hyperactivity of the parathyroid glands.

Critical analysis of the literature leads us to the single conclusion that parathyroid hyperactivity should be suspected only if there are a negative calcium and a negative phosphorus balance and an increased phosphatase content, associated with roentgenographic findings in the skeleton characteristic of osteitis fibrosa cystica. In similar conditions the clinical picture may show various features, but the underlying disorder must give evidence of the same cardinal characteristics. The osteoporosis encountered in different conditions, especially that associated with blue scleras, is an entirely different pathologic entity, not based on disturbances of the parathyroid glands.

CHANGES IN THE BONE ASSOCIATED WITH BLUE SCLERAS

The skeletal changes in connection with blue scleras are described as osteopsathyrosis or osteogenesis imperfecta. Many authors are of the opinion that these are merely two aspects of the same condition, both showing evidence of deficient osteoblastic function. Looser, therefore, made the suggestion that "the essential unity of the two conditions should be recognized by giving osteopsathyrosis the name of osteogenesis imperfecta tarda." Bauer took an identical stand, affirming the existence of embryologic defectiveness.

According to Bauer, osteogenesis imperfecta is a condition involving the connective tissue as a whole, manifesting itself as a dysfunction of all the matrix-forming cells. While all tissues of mesenchymal origin are affected, the more highly organized ones (bone, cartilage and tooth pulp) are chiefly concerned in the disease process. That the cell formation is not necessarily deficient is exemplified by the case reported by Buday, in which osteoblasts were present in great numbers and exhibited normal structure but their matrix-forming function was impaired. Fahr and Krauss failed to confirm Bauer's theory of dysfunction of the matrix-forming cells. Fahr reported excessive development of the thyroid and thymus (which are derived from entoderm) and of the pituitary and adrenal medulla (an ectodermal derivative), but his objections were based mainly on a singular unconfirmed case.

Various observers have described changes in other tissues of mesodermal origin, such as muscular hypotonia and an excess of lymphocytes in the blood (Frontali, Macciotta). Other authors, differing with Bauer, pointed out involvement of tissue of epiblastic origin (enamel, the auditory nerve). As a result mesenchymal deficiency is not generally accepted as a true and sufficient explanation of the osseous changes associated with blue scleras. Many of the recent authors agreed that the lack of osteoblasts (due to dysfunction, according to Bauer) is the principal characteristic in the histologic picture of osteogenesis imperfecta in the syndrome of blue scleras.

The question then arises as to whether the skeletal defectiveness present might be traceable to an original malformation, or vitium primae formationis (Kaufman, Dicherle, Sumita), or to disturbances influencing the bone-forming tissues only secondarily. The most exhaustive microscopic study was made by Weber,¹⁹ in the case of a girl 2 days old with early and recent fractures of the long bones and ribs (with no mention of blue scleras). This case demonstrated that osteogenesis imperfecta is a generalized disorder affecting similarly the entire bone-building system and that its origin is traceable to an early stage of intra-uterine life.

According to Weber, the picture of osteogenesis imperfecta is one of arrested development with active and passive manifestations. The passive manifestation of arrested development is the lack of the highest unit, the osteon. Either no osteoblasts are present, or they are unable to perform their function—the production of shell bone. This arrestment in the destined development is primary, so that the formation of lamellar bone never occurs.

The active manifestation is encountered in the bone tissue actually formed, the entire skeleton consisting of imperfect fiber bone. The latter is ontogenetically and phylogenetically the oldest type of bone tissue, forming the nucleus for subsequent deposition of the shell bone. Thus in osteogenesis imperfecta the formation of bone tissue is arrested in the stage of nucleus formation.

Weber described the pathologic changes of osteogenesis imperfecta as a relative shift to the left. If an attempt were made to present the separate stages of bone formation graphically, the highest one on the extreme right would be lacking. In the middle stages bone formation would be seemingly increased (whereas it is only relatively increased); that is, the fiber bone is formed in normal quantity but is not resorbed by the osteoblasts, owing to their absence. In the lowest stage bone formation would be less than normal. An important sign is the inferior

19. Weber, M.: Osteogenesis Imperfecta Congenita, Arch. Path. 9:984 (May) 1930.

quality of the intercellular substance in the fiber bone. Since this pathologic fiber bone has its origin in embryonal tissue, the conclusion is confirmed that this condition is a congenital malformation.

Weber agreed with Kaufman and Bauer in assuming a germinal and not a secondary disturbance, for in osteogenesis imperfecta the osteoblasts and giant cells either are lacking or are failing in function. Both these cellular conditions originate in the endothelial cells of the blood vessels, and thus the latter are responsible for the changes in the bone formation.

The occurrence of severe arteriosclerosis in some cases of osteogenesis imperfecta also points to vascular involvement. The juvenile type of arteriosclerosis indicates a disturbance in the composition of intercellular substances similar to that in the pathologic formation of fiber bone. Thus the changes are traceable as far back as mesenchymal development, and the analysis and minute observation made by Bauer on mesenchymal dysfunction are confirmed.

METABOLIC CHANGES IN CASES OF BLUE SCLERAS

The pathologic changes in osteogenesis imperfecta and osteitis fibrosa cystica are, therefore, well characterized and well defined. It is easily perceived that the underlying pathologic picture of each is absolutely different. Evidently, the changes in the calcium and the phosphorus metabolism in osteogenesis imperfecta associated with blue scleras should vary from the usual negative balance in cases of osteitis fibrosa cystica, which is the clinical manifestation of osseous changes in hyperactivity of the parathyroid glands.

Bookman,²⁰ without mentioning blue scleras, reported that in cases of active osteogenesis imperfecta the retention of calcium is somewhat or decidedly below normal. Similar statements relative to a retention of calcium lower than normal were made by Schabad.²¹ In a child 7 months old with this osseous condition Schwarz and Bass²² found a positive calcium balance, a retention of calcium somewhat below normal and a practically normal phosphorus metabolism; no reference to blue scleras was made. According to af Klercker,²³ in this condition the retention of calcium is normal but at its lower range, while Flagstad, Zanger and Leven²⁴ cited a normal concentration of both calcium and phosphorus.

20. Bookman, A.: The Metabolism in Osteogenesis Imperfecta with Special Reference to Calcium, *Am. J. Dis. Child.* **7**:436 (June) 1914.

21. Schabad, I. A.: *Ztschr. f. Kinderh.* **11**:230, 1914.

22. Schwarz, H., and Bass, M. H.: Osteogenesis Imperfecta: Report of a Case with the Study of Its Metabolism, *Am. J. Dis. Child.* **5**:131 (Feb.) 1913.

23. af Klercker, K. O.: *Monatschr. f. Kinderh.* **25**:338, 1923.

24. Flagstad, A. E.; Zanger, E., and Leven, L.: *Minnesota Med.* **7**:800, 1924.

In Hunter's²⁵ typical case a boy aged 17, with an irrelevant family history, had blue scleras, associated with multiple fractures (more than thirty), which began at the age of 3 weeks. Hunter considered the negative calcium balance as due to the low intake, for in the control case the determination was identical, and he therefore concluded there was no increased loss of calcium. Tauber²⁶ observed two sisters, the older of whom showed marked osteogenesis imperfecta while the younger showed only traces of this disease. In both cases the negative calcium balance was improved by the use of different therapeutic measures. Sindler's²⁷ patient, a 20 day old child, showed a serum calcium content of 7.8 mg. per hundred cubic centimeters and a normal calcium metabolism. Two patients examined by Holler²⁸ had blue scleras and multiple fractures and showed a blood calcium content of 0.015 per cent, estimated by the Jansen method—a slight increase above the normal (0.01 per cent). Wyatt and McEachern's²⁹ patient, an infant 4½ weeks old, had a serum calcium content of 10.2 mg. and a phosphorus content of 6.5 mg. per hundred cubic centimeters, the calcium and phosphorus contents of bone ash and their ratio being normal. In Kay's³⁰ opinion, there is a tendency to higher values than normal for calcium and phosphorus in fragilitas ossium but no constant increase of phosphatase. The average value for phosphatase in four cases was 0.5 unit, the normal for the age group being 0.22 unit. In some cases of osteitis fibrosa generalisata, osteitis deformans, osteomalacia and rickets the value for phosphatase rises above 3 units, which is more than twenty times the normal average.

Two patients with osteogenesis imperfecta were observed by Hassman and Verdino.³¹ The first showed higher values than normal for calcium and normal values for phosphorus in the whole blood and an increased amount of phosphorus in the serum. The second patient showed a similar condition, but in this case the serum phosphorus content was normal. In their observation of the disease Bodansky and Jaffe³² found a normal average for the phosphatase content in the blood serum—about 2.7 and 8 units per hundred cubic centimeters in adults and children, respectively—but a greatly increased value in polyostotic Paget's disease (fifty times greater than normal), in rickets (twenty

25. Hunter, D.: *Lancet* 1:9, 1927.

26. Tauber, M.: *Monatschr. f. Kinderh.* 36:12, 1927.

27. Sindler, A.: *Ztschr. f. Kinderh.* 42:85, 1926.

28. Holler: *Verhandl. d. deutsch. orthop. Gesellsch.* 20:273, 1925.

29. Wyatt, T. C., and McEachern, T. H.: Congenital Bone Dysplasia (Osteogenesis Imperfecta) Associated with Lesions of the Parathyroid Glands, *Am. J. Dis. Child.* 43:403 (Feb.) 1932.

30. Kay, H. D.: *J. Biol. Chem.* 89:249, 1930.

31. Hassman, K., and Verdino, A.: *Jahrb. f. Kinderh.* 140:1, 1933.

32. Bodansky, A., and Jaffe, H. L.: *J. Biol. Chem.* 105:11, 1934.

times greater than normal) and in hyperthyroidism (ten times greater than normal), a lower increase in generalized osteoporosis and osteomalacia and a similar but inconstant content in fragilitas ossium.

In four cases of osteogenesis imperfecta Hansen³³ observed values for phosphatase almost identical with those in the control cases of subjects of the same age group. Aub and Farquharson³⁴ reported one case of fragilitas ossium not associated with blue scleras, in which repeated fractures occurred from the slightest trauma, the roentgenograms showing osteomalacia. The excretion of calcium with a low calcium diet was normal, although the urinary output of calcium is probably a little higher than normal in most children of the patient's age. The calcium content of the blood serum was normal; the phosphorus content was slightly above the usual level for children.

Takahashi reported four cases of blue scleras in which there was an increased urinary output of calcium as compared to the normal output of calcium in three other cases. In his study the calcium balance was not established. The calcium level in the blood was examined in only one of the four cases, and a calcium content of 5.6 mg. per hundred cubic centimeters was reported, in contrast to the normal calcium levels of two controls (11.43 mg. and 9.97 mg.). This hypocalcemia led him to conclude that there was hypo-activity of the parathyroid glands, an assertion which was generally adopted and which was quoted many times in the literature. The validity of such an assertion must not be too hastily accepted, particularly since many other authors, using the same technic, could not establish such a low calcium level in the blood. Paal,³⁵ in his first case, found 9.066 mg.; in the second case, 9.99 and 9.73 mg., and in the third, 12.53 and 12.8 mg., the latter values being somewhat pathologically increased. The author remarked that in the last case the roentgen ray translucency was not typical, while it was typical in the two former cases. Stevenson and Cuthbertson,³⁶ examining the calcium and phosphorus levels in four cases in which the intake of calcium was controlled, found these to be within the normal limits in all instances. Examination of the urine and feces for the retention of calcium oxide (CaO) and phosphorus pentoxide (P₂O₅) showed a diminished amount in two cases (in one the retention of magnesium was also much diminished) and an almost normal content in the third case. In the fourth case the metabolic investigation was made eleven weeks after a serious accident resulting in multiple fractures and revealed a decidedly diminished retention. At that time there was still

33. Hansen, A. E.: *Proc. Soc. Exper. Biol. & Med.* **31**:1023, 1934.

34. Aub, J. C., and Farquharson, R. F.: *J. Clin. Investigation* **11**:235, 1932.

35. Paal, H.: *Klin. Wchnschr.* **8**:1304, 1929.

36. Stevenson, G. H., and Cuthbertson, D. P.: *Lancet* **2**:782, 1931.

absence of callus formation. A few weeks later, when the callus had formed, the retention of calcium oxide and phosphorus pentoxide became less marked.

In a typical instance of osteogenesis imperfecta and blue scleras, in which ten days after birth a fracture of the humerus was present and during the ensuing ten years numerous spontaneous fractures were sustained, the blood calcium level, as determined repeatedly by Gyllenswärd,³⁷ was 8.6, 9.6 and 9.9 mg. per hundred cubic centimeters. Ottley³⁸ reported 10.2 mg. of calcium in the blood serum. Hirschman in his first case found 13.06 mg. and in the second 16 mg. These rather high levels, in his opinion, did not indicate any endocrine involvement. Unfortunately, Hirschman failed to establish the calcium balance in his cases. As most of these authors omitted the value for phosphatase in all these metabolic determinations, their figures are not enlightening.

Kleinberg³⁹ recently reported two cases of osteogenesis imperfecta, with accurate laboratory findings. The first patient, a 2 month old child, who had exhibited fractures at birth, had a blood calcium content of 10.3 mg., a blood phosphorus content of 5.6 mg. and a phosphatase content of 15.6 units. The second patient, a 2 year old child with deep blue scleras (and proof of blue scleras in the antecedents) showed normal values for calcium, phosphorus and phosphatase. The blood calcium content amounted to 10.5 mg. in the first case reported by Harnett⁴⁰ and to 10 mg. in the second case (in which the mother, brother and sister also exhibited blue scleras).

It is evident that there is a lack of uniformity in the laboratory determinations and findings in the cases of blue scleras so far reported. In order to elaborate on the laboratory data in cases of osteogenesis imperfecta we report two cases which we had an opportunity to observe in the wards of the Beth Israel Hospital. Not only were the examinations of the serum calcium accurately made, but the calcium metabolism was carefully studied. To add to the latter study, the values for phosphorus and phosphatase were also determined, and roentgenographic examinations were made.

REPORT OF CASES

CASE 1.—*History*.—A white boy, 12 years old, with markedly blue scleras since birth complained chiefly of inability to walk and the past occurrence of repeated fractures on sustaining trivial injury. The fractures were as follows: (1) fracture of the right clavicle, which was sustained when he was being lifted out of a carriage at the age of 5 months; (2) fracture of the right clavicle again

37. Gyllenswärd, C.: *Acta pædiat.* **13**:111, 1932.

38. Ottley, C. M.: *Arch. Dis. Childhood* **7**:137, 1934.

39. Kleinberg, S.: *J. Bone & Joint Surg.* **16**:953, 1934.

40. Harnett, W. L.: *Brit. J. Surg.* **22**:269, 1934.

when he was 2 years old; (3) fracture of the left wrist at 7 years of age; (4) fracture of the left wrist a second time, at 8 years of age, and (5) fracture of the left femur at 10 years of age.

All the fractures healed normally. The patient was able to walk, but with difficulty, until the left femur was fractured, when the use of crutches became necessary. In addition he had congenital clubfoot.

Family History.—No other members of the family had any osseous dyscrasias.

Physical Examination.—The boy was well developed and obese, measuring 5 feet and 2 inches (157.5 cm.) in height and weighing 125 pounds (56.7 Kg.). The blood pressure was 120 systolic and 88 diastolic.

Head: The occipitofrontal circumference was 22 inches (55.8 cm.). There was bitemporal bulging.

Eyes: Both scleras were deep china blue, especially in a ring about 0.5 cm. around the limbus. Toward the equator the color become less intensive. The pupils were equal and regular and reacted well to light and in accommodation. The fundi were normal. The conjunctivae showed a good hemic component.

Nose, Mouth and Teeth: These were normal.

Chest: On inspiration the circumference measured 34 inches (86.4 cm.); on expiration it measured 33½ inches (85.2 cm.). Both breasts were large and obese; no fluid could be expressed.

Heart: This organ was not enlarged. The sounds were regular and of good quality. The pulmonic second sound was greater than the aortic second sound.

Extremities: These were obese and hypotonic. The wrists were double-jointed. There was a definite weakness in the grasp of the hands, especially of the left hand, and a weakness of the extensor muscles of the thighs. There was a tendency to bilateral foot drop, although the feet could be flexed. The right foot was held in an abductor position from the midline, and the left foot was held in adduction (toward the midline). There was some tendency toward knock knee (genu valgum). The patient could not raise his legs (to put on stockings) without the support of his arms.

The fingers were long, thin and tapering, with lateral curvature of the nails and a slight cyanotic hue. The little finger of the left hand was deformed, owing to a lateral curvature of the first two phalanges (caused by an old fracture).

Measurements: Both the left and the right biceps measured 10 inches (25.4 cm.). The distance from the acromion process of the clavicle to the inner condyle of the ulna was 21 inches (53.3 cm.) on both the right and the left. The midportion of the thigh measured 17 inches (43.2 cm.) on both the right and the left. The lower third of both tibias measured 11½ inches (29.2 cm.). The distance from the left anterior-superior spine to the interior malleolus on the right was 33¾ inches (85.7 cm.) and to that on the left, 35 inches (88.9 cm.). A scar 3 inches (7.6 cm.) long was present on the inner aspect of the right foot and above the right heel, and another scar, 5½ inches (13.9 cm.) long, was seen on the lower half (inner aspect) of the right leg. The scar formation was due to previously performed bone graft from the right tibia into the right os naviculare. No callus formation was present.

There were bowing and thickening of the distal third of the left radius anteriorly, and a small dorsal deformity was felt in the wrist of the left hand.

Reflexes: The knee jerks were present and equal. The biceps reflexes were present but sluggish. No pathologic reflexes were elicited.

Posture: Slight scoliosis (an old spinal curvature corrected by a brace and the application of a cast) was present.

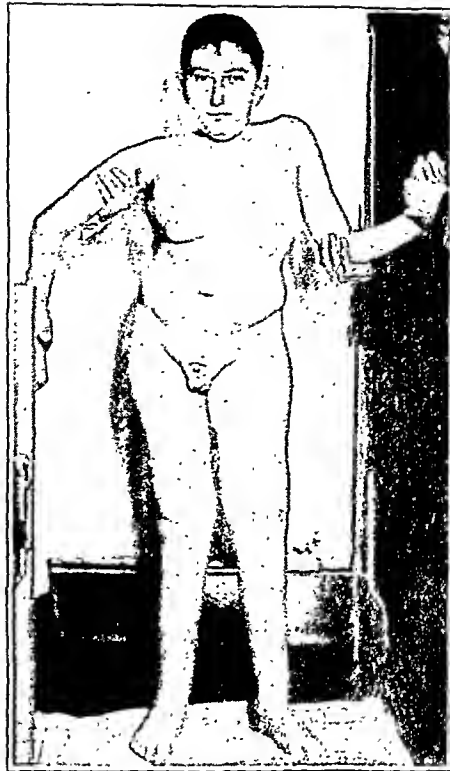


Fig. 1 (case 1).—Clinical appearance of patient. Owing to the multiple fractures suffered at various periods, he has to be supported.



Fig. 2 (case 1).—Roentgenogram of the knees of patient. The osteoporosis is extremely marked, and the decalcification of all the bones is pronounced. The shaft of the left tibia shows evidence of old fractures.

Joints: These were lax, indicating hypotonicity of the ligaments and tendons. There was hyperextension of the fingers at the interphalangeal, metacarpophalangeal and wrist joints. When the hand was held outward it fell as in wrist drop. At the elbows there were hyperextension and lateral motion, indicating ligamentous weakness.

There was undue mobility at the ankle joint, the lack of muscular resistance being striking. The same laxity was noted at the knee joint, with increased lateral mobility and a little rotary movement.

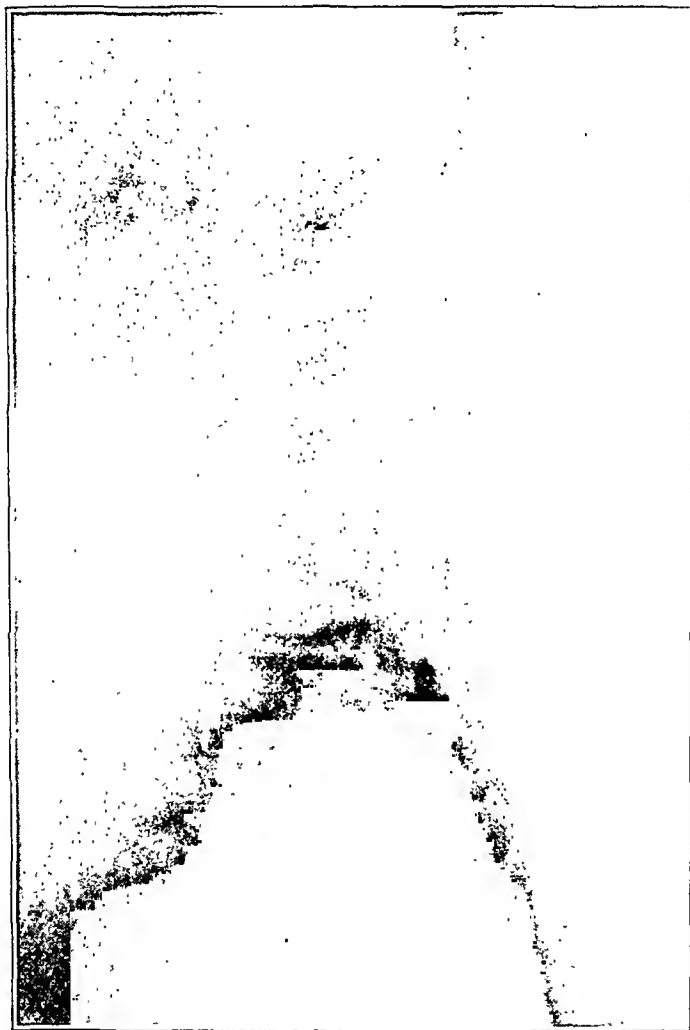


Fig. 3 (case 1).—Roentgenogram of the spinal column of patient, showing highly marked osteoporosis of all the vertebrae.

On palpation the muscles were distinctly hypotonic.

Neurologic Examination (Dr. L. Loeser).—The minimum response of the right facial nerve to the galvanic current was anodal closure contraction with 4 milliamperes and cathodal closure contraction with 5 milliamperes. The minimum response of the right ulnar nerve was anodal closure contraction with 3 milliamperes and cathodal closure contraction with 4 milliamperes. The minimum response of the right deep peroneal nerve (flexion of the foot) was anodal closure

contraction and cathodal closure contraction with 6 milliamperes (with much more forcible and greater contraction of the muscles of the foot on cathodal closure contraction than on anodal closure contraction, showing qualitatively the absence of degeneration of the nerve). The reactions indicated diminished muscular contractility due not to nerve degeneration but to muscle degeneration.

Roentgenographic Findings (Dr. J. Furst).—The sella turcica was comparatively small in the proportional transverse dimension (50 per cent undersized). The skull was of the ovoid type. The first anterior rib on both sides showed a moderate degree of porosity and a tendency to flaring. The extremities showed a marked degree of osteoporosis. This was especially observed at the cancellous ends of the long bones. The variation in structural detail at the lower end of the left femur was probably the end-result of a healed fracture at this point. A similar condition was observed at the intermediate third of the shaft of the right tibia.

The tibia and fibula of both sides showed a tendency to bowing.

The entire dorsal and lumbar parts of the spine showed a marked loss of calcium and poor definition of the medullary structure. In the lateral view the bodies of the vertebrae were concave. The fourth lumbar vertebra was almost in collapse. The dorsal part of the spine showed a moderate posterior curvature.

Laboratory Findings.—The urine was clear and had a specific gravity of 1.006. A blood count showed the following values: 71 per cent hemoglobin; 4,630,000 erythrocytes, and 8,000 leukocytes, with 50 per cent polymorphonuclears, 36 per cent lymphocytes, 5 per cent endothelial leukocytes, 6 per cent eosinophils, and 3 per cent basophils. There were slight anisocytosis and very slight poikilocytosis. The Wassermann, Kline and Kahn tests of the blood gave negative results. The calcium content of the blood was 10.1 mg. per hundred cubic centimeters; the phosphorus content was 3.8 mg., and the phosphatase content, 3.9 units (1 unit = 1 mg. of phosphatase liberated in one hour).

Calcium Balance (Six Day Test): The patient was kept on a special diet consisting of 60 Gm. of proteins, 33.5 Gm. of fats and 250 Gm. of carbohydrates. The daily intake of calcium was 0.121 Gm. Three glasses of water and 1 Gm. of sodium chloride were allowed daily. Carmine was used in order to delimit the beginning and end of the test. The calcium content was determined by the Kramer-Tisdall method; the phosphorus content, by the Benedict method. The results were as follows:

	Calcium, Gm. per 100 Cc.	Phosphorus Pentoxide, Gm. per 100 Cc.	Chlorides, Gm. per 100 Cc.
Urine.....	0.0933	5.496	23.1410
Feces.....	0.4980	0.251	0.0518
Total.....	0.5913	5.747	23.1928
Food.....	0.9540	1.261	2.5740

CASE 2.—History.—D. O., a white boy aged 15 months, had broken bones frequently, without traumatism. He was born spontaneously at home. He cried almost continuously as though in pain. His mother noticed that his legs were swollen. A physician informed her that the baby's legs and hips were fractured. The patient was kept in the hospital for six weeks, after which time the fractures were healed. Three or four months later a slight trauma produced a fracture of the left leg. In six weeks this leg healed. One or two months later, while the baby was being dressed, the left leg snapped again. This fracture healed well. Ten weeks before the patient was brought for examination, the mother, after carrying him a long distance, placed him gently on a counter, and the right leg broke. Three days later the patient suffered another fracture of the leg.

His appetite was poor, and there had been no regular gain in weight. There were two other children, both of whom were well and without any indication of a similar condition.

Physical Examination.—There was a definite deformity of the head, as though in passage through the birth canal there had been pressure in the frontal and occipital regions. There were dilated venules on the prominent forehead. The anterior fontanel admitted two fingers and was not bulging. The posterior fontanel was closed. There was loss of some hair in the occipital region. The head was enlarged, having an occipitofrontal circumference of 18 inches (45.7 cm.).

Eyes: The scleras showed definite but slight blue discoloration of equal hue. The lower lids were wrinkled.



Fig. 4 (case 2).—Clinical appearance of patient.

Mouth: The patient had a high-arched palate. Six upper and six lower teeth were present (delayed dentition). They were poorly developed and appeared decalcified.

Extremities: The right leg was shorter than the left. The distance from the anterior-superior spine to the internal malleolus measured $10\frac{1}{2}$ inches (26.6 cm.) on the right side and 10 inches (25.4 cm.) on the left. The midsection of the right thigh measured 7 inches (17.8 cm.), and that of the left thigh, 8 inches (20.3 cm.). The left thigh showed anterior bowing and thickening.

Röntgenographic Findings (Dr. J. Furst).—Examination of the skull showed it to be of the hydrocephalic type and somewhat ovoid. The greatest increase in the skull's diameter was in the vertical oblique direction.

The upper extremities showed a moderate degree of absorption of lime salts, which had resulted in accentuation of the medulla and sharpening of the cortices, which probably contained increased amounts of lime salts.

The lower extremities showed a similar general appearance of the bones. There was evidence of old fractures of both femurs with a slight increase in their external bowing. More recent fractures were observed in the shaft of the tibia.

These findings suggested a systemic disturbance—osteoporosis imperfecta.

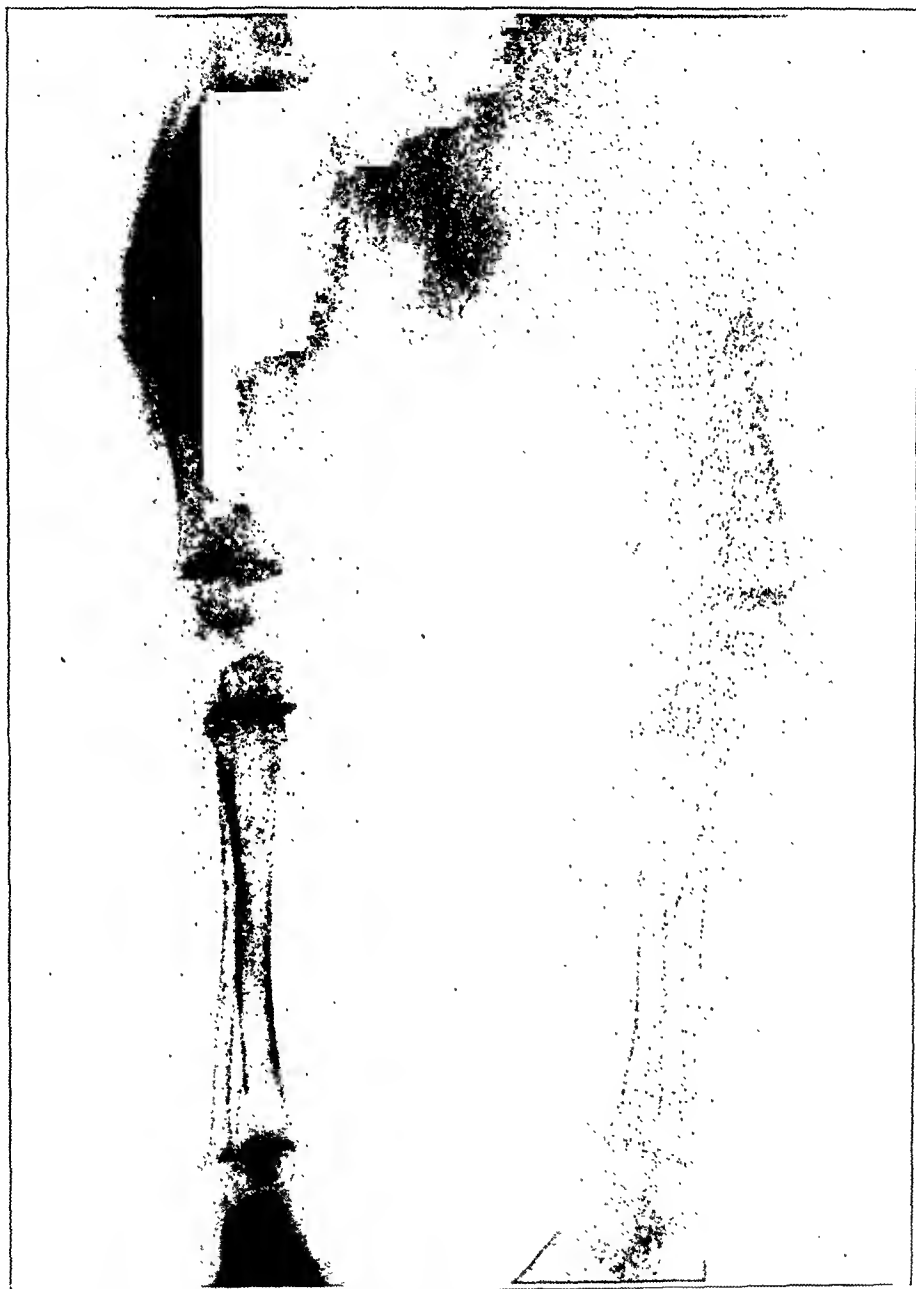


Fig. 5 (case 2).—Roentgenogram of the lower extremities of patient. In addition to generalized osteoporosis, evidence of eight new and old fractures is visible.

Laboratory Findings.—The urine was cloudy, with an acid reaction; the specific gravity was 1.024. The Wassermann, Kline and Kahn tests of the blood gave negative results. The calcium content of the blood was 11 mg. per hundred cubic centimeters; the phosphorus content was 3.4 mg. Two weeks later a second examination showed the calcium content to be 11.1 mg. and the phosphorus

content 4.7 mg. After another interval of two weeks the phosphorus content was 3.5 mg., and the phosphatase content, 4.9 units.

The calcium balance was estimated by the same methods as in case 1. The results were as follows:

Test of First Three Days			
	Calcium, Gm. per 100 Ce.	Phosphorus Pentoxide, Gm. per 100 Ce.	Chlorides, Gm. per 100 Ce.
Urine.....	0.011	0.408	0.5590
Feces.....	0.211	0.019	0.0743
Total.....	0.222	0.457	0.6333
Food.....	0.295	0.233	0.0618
Test of Second Three Days			
Urine.....	0.023	1.249	1.537
Feces.....	0.617	0.193	0.289
Total.....	0.642	1.442	1.826
Food.....	1.749	0.550	1.734

DIFFERENTIAL FACTORS

Our metabolic study strongly confirms the decided differences in the pathologic osseous conditions existing in hyperparathyroidism and those associated with blue scleras. Hyperactivity of the parathyroid glands results in a negative calcium and a negative phosphorus balance, with increased activity of phosphatase. This is in contradistinction to the findings in osteogenesis imperfecta, in which there is no hypercalcemia or hypophosphatemia and no increased excretion of calcium and phosphorus through the urine.

A careful perusal of the literature shows, furthermore, that in the cases of osteogenesis imperfecta and associated blue scleras in which an endocrine disturbance was assumed this assumption was based only on vague clinical symptoms, without consideration of the extremely important metabolic data and accurate investigation of the pathologic changes in the bones. Accordingly, various endocrine glands were suspected of being the causative factor. Kaznelson held the parathyroids responsible; Lesné, Hutinel and Dreyfus-Sée, and Sverdlov,⁴¹ the thyroid, and Gutzeit, the hypophysis. Frocher mentioned changes in the anterior lobe of the pituitary as a direct cause and Schwarz, changes in the thymus, while Hein described an enlarged thymus. Casanovas pointed to hypofunction of the parathyroid glands or the thyroid, and Viallefont⁴² and Terrien, to the parathyroid glands. On the other hand, Desoff expressed the opinion that the rather well established presence of hyperparathyroidism must be the source of the syndrome. These rather speculative assertions are, to say the least, not helpful in clarifying the relationship of the two conditions and are even misleading not only in the classification of the anomaly but in regard to therapeutic measures for, and operative intervention on, the suspected glands as well.

41. Sverdlov, G. O.: *Zentralbl. f. d. ges. Ophth.* **28**:75, 1933.

42. Viallefont, H.: *Ann. d'ocul.* **170**:289, 1933.

In considering both the similarities and the differences in the two conditions it should be borne in mind that osteogenesis imperfecta associated with blue scleras is characterized by dominant hereditary transmission accompanied by deafness. Two of the symptoms, the blue scleras and the fragile bones, are present at birth. Osteitis fibrosa cystica is likewise a generalized disease of the entire skeleton, leading to numerous spontaneous fractures, but appears at a much later stage of life. Hunter and Turnbull considered the usual age for its appearance to be from 30 to 35 years. Gutman, Swenson and Parsons,⁴³ in analyzing one hundred and fifteen cases of hyperparathyroidism according to the patients' ages at the time of operation or death, found no patients in the first decade of life, seven in the second, seventeen in the third, nineteen in the fourth, twenty-eight in the fifth, twenty-three in the sixth and five in the seventh. Age is therefore an important clinical factor in the evaluation of the causes of spontaneous fractures. In osteogenesis imperfecta the fractures are intra-uterine or acquired in early life, while in osteitis fibrosa cystica they occur at a later age, a period when in the former condition no further pathologic changes take place.

In the course of osteogenesis imperfecta hereditary deafness is likely to develop after the age of 20, being caused, according to the roentgenographic findings of Stenvers,⁴⁴ by structural changes in the petrous bone and by calcification of the labyrinth. The pathologic osseous changes in osteogenesis imperfecta were outlined by Albright, Aub and Bauer^{15a} as a depression in bone formation combined with normal absorption, with no increase in osteoclasts or in fibrosis. It is well to point out again that this disease is due to congenital faulty differentiation or malformation of the mesenchyma, owing to which the osteoblasts either are lacking or are unable to perform their allotted physiologic function, the end-result being the formation of fibrous bone tissue instead of further development into shell bone. There is no active mobilization of the calcium supply in the body, and consequently there is no negative calcium or phosphorus balance and no increase in phosphatase activity. The spontaneous multiple fractures and the prompt healing without considerable callus formation manifest themselves in both conditions but are based on entirely different pathologic principles. In contrast to the observations in cases of osteogenesis imperfecta, in those of osteitis fibrosa cystica there are typical laboratory findings showing the active mobilization of calcium and phosphorus. This results in the unique combination of a negative balance of these compounds

43. Gutman, A. B.; Swenson, P. C., and Parsons, W. B.: The Differential Diagnosis of Hyperparathyroidism, *J. A. M. A.* **103**:87 (July 14) 1934.

44. Stenvers, H. W.: *Arch. f. Ophthl.* **95**:94, 1918.

with an increased blood phosphatase content, changes which are caused by overproduction of the active principle of the parathyroid glands. The metabolic alterations are usually marked and show a dramatic change following removal of the tumor almost invariably found in hyperparathyroidism, with associated elimination of the excessive stimulus of osteoclastic resorption.

The underlying cause of the excessive excretion of calcium and phosphorus is generally adenoma of the parathyroid glands (the adenoma being benign only in relation to its morphology). Some authors, like Gold, prefer to follow the classification of struma instead of tumors and use the term *struma parathyroidea diffusa* or *nodosa parenchymatosa*.

The adenomas are of minute size and show increased vascularization and decrease in, or absence of, the fat cells. The principal cells are mostly considered as functionally essential to the secretion of the gland, but opinions differ widely as to which types of the chief cells are the important ones. Petersen and Schall considered the less chromatic types as important; Getzova and Hartwich, the darker red ones. On the other hand, Koenigstein and Forsyth regarded the oxyphil cells as the active and colloid-producing types. The number of cells seems to increase with the age of the patient, a factor probably associated with the amount of colloid and fat deposited. Some observers even entertained the idea that the principal and oxyphil cells are possibly only different morphologic modifications representing various stages of the physiologic function. In their series of instances in which a single adenoma occurred Albright, Bloomberg, Castelman and Churchill^{15c} noted that the tumor consisted of slightly enlarged hyperchromatic chief cells without mitosis, the cells being arranged in columnar or cylindric formation. In addition to these, cells with water-clear cytoplasm (Getzova) and oxyphil cells were present, the former rarely comprising the major part of the specimen.

The single adenoma or, possibly, multiple adenomas of the gland are doubtlessly responsible for the pathologic picture of parathyroid hyperactivity. Rarely, however, does one encounter cases with the classic metabolic findings of hyperparathyroidism in which, instead of adenomas, hyperplasia of all the parathyroid tissues is common. When hyperplasia is present, according to Albright and his co-workers, the water-clear cells predominate; they are of a much larger size than those encountered in the single tumors. The hyperplasia of the thyroid glands, in conjunction with the metabolic picture of hyperparathyroidism, is considered as forming an important entity and suggests the pituitary gland as the source of stimulation. This pathologic picture must be borne in mind in order to differentiate this form of hyperparathyroidism from the "hypertrophy of Erdheim" of the parathyroid glands in condi-

tions such as osteomalacia and rickets. In the latter, hyperplasia represents an inadequate attempt at compensation, the alteration being the result and not the cause of the disease. Primarily, hyperfunction and dysfunction are established in the sense of regulatory function, which induces hyperplasia of a correlative regulatory character and by over-taxation causes pathogenic action on the skeleton.

PRESENCE OF BLUE SCLERAS IN HYPERPARATHYROIDISM

A study of the literature of known and proved cases of hyperparathyroidism (associated with adenomas and hyperplasias) yields little about blue scleras. In more than one hundred cases we could find only four instances in which blue scleras were noticed in connection with distinct parathyroid hyperactivity. These cases are briefly summarized.

Wichmann⁴⁵ reported a typical case of a woman 45 years of age on whom an exact determination of the calcium metabolism was made. The scleras were described as mildly bluish. In the first of two cases of parathyroid tumor cited by Hanke,⁴⁶ a woman aged 33 and her mother both showed blue scleras. The subjective complaints began at the age of 18, and she had her first spontaneous fracture at the age of 27. The patient exhibited typical hyperparathyroidism; operation proved the presence of adenomas. Similarly, Cohen and Kelly⁴⁷ reported the case of a woman 48 years of age who showed swelling of the jaw at the age of 39, associated with blue scleras. She presented the clinical picture of hyperparathyroidism—a negative calcium balance and a tumor of the right inferior parathyroid gland. Keynes and Taylor⁴⁸ noticed brilliantly blue scleras in a man who suffered from osteoclastoma of the right maxilla, muscular atony, spontaneous fracture of the right humerus and generalized typical pathologic osseous changes caused by a large tumor of the left parathyroid gland. In all these four cases the presence of blue scleras was observed. In Hanke's case even hereditary transmission was mentioned.

It would be tedious to enumerate all the classic symptoms of parathyroid hyperactivity in these four cases, as they were numerous. Suffice it to say that blue scleras were present in intimate association with typical parathyroid disorder. We must emphasize, however, that the clinical picture, the occurrence of fractures at a rather later period of life, is more characteristic of parathyroid disorder than of the symptom complex of osteogenesis imperfecta. These instances stand in marked contradistinction to the occurrence of blue scleras with osteoporosis and

45. Wichmann, F. W.: *Deutsche Ztschr. f. Chir.* **235**:619, 1932.

46. Hanke, H.: *Arch. f. klin. Chir.* **172**:366, 1932.

47. Cohen, H., and Kelly, R. E.: *Brit. J. Surg.* **20**:472, 1933.

48. Keynes, G., and Taylor, H.: *Brit. J. Surg.* **21**:20, 1933.

deafness but with absence of parathyroid involvement. Our two cases herein reported, in which the calcium and phosphorus balances were carefully determined, illustrate the fact clearly.

On the basis of the present-day knowledge, an explanation of the simultaneous presence of hyperparathyroidism and blue scleras in the four reported cases is still lacking. There is the possibility of the coexistence of two different pathologic entities, a congenital syndrome of blue scleras and superimposed parathyroid tumor formation, causing the typical negative calcium and negative phosphorus balance and the pathologic osseous condition, which are never encountered together in association with the congenital form of blue scleras alone.

As an item of interest mention may be made of Albers-Schönberg's disease, or osteopetrosis, which roentgenographically and pathologically may be regarded as diametrically different from the osteoporosis associated with blue scleras. In cases of marble bones there are a striking familial tendency and, occasionally, a hereditary influence. McCune and Bradley⁴⁹ thought that osteopetrosis is a true developmental disease traceable to faulty differentiation of the primitive common forerunner of osteogenic and hematogenic tissue, the ultimate cause being inherent in the germ plasm. There is no evidence at present to suggest any endocrine disturbance. The inconstant changes in the calcium and the phosphorus metabolism that occur occasionally in cases of osteogenesis imperfecta and osteopetrosis are probably only secondary disturbances due to the primary disorder of the bone formation in this exceedingly rare anomaly.

CONCLUSIONS

In the usual cases of blue scleras associated with spontaneous fractures and hereditary deafness there are not sufficient variations in the values for calcium, phosphorus and phosphatase to warrant a supposition of involved endocrine disturbance.

In osteogenesis imperfecta there is evidence of decided hereditary transmission, in contrast to hyperparathyroidism, which shows no such influence.

Osteitis fibrosa cystica is based on hyperactivity of the parathyroid glands, the usual cause being tumor formation within the glands, resulting in mobilization of the supply of calcium. The spontaneous fractures occur in adult life and are accompanied by a negative calcium balance, cyst formation and giant cell tumors of the bones.

The small minority of cases in which blue scleras are associated with proved parathyroid disorder are rare exceptions, the pathologic

49. McCune, D. J., and Bradley, C.: Osteopetrosis (Marble Bones) in an Infant; Review of the Literature and Report of a Case, *Am. J. Dis. Child.* **48**:949 (Nov.) 1934.

features not constituting the usual picture. The most plausible explanation lies in the possibility that the condition of hyperparathyroidism has been superimposed on a preexistent congenital anomaly.

Similarly, spontaneous fractures are clinical features of the other form of generalized osseous disease, osteogenesis imperfecta, associated with blue scleras and deafness. They occur in utero or in infancy or childhood, in contradistinction to the adult age incidence of hyperparathyroidism. The roentgenographic picture of the osseous condition is that of osteoporosis. There are absence of a negative calcium balance and lack of increased phosphatase activity in osteogenesis imperfecta, the latter being a distinguishing feature of the parathyroid syndrome. Furthermore, blue scleras are characterized by dominant hereditary transmission and are due to a congenital faulty differentiation or malformation of the mesenchyma.

PARTIAL RUPTURE OF THE LAMINA CRIBROSA FROM CONTUSION OF THE EYEBALL

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Injury of the optic nerve in the scleral canal is rare and usually results from a penetrating wound of the eye (Parsons¹). Clinical cases of indirect rupture of the lamina cribrosa have been recorded by Lang,² Cramer³ and Gonin.⁴ As the eyeball was not removed in these cases, microscopic descriptions are lacking. Wagenmann⁵ did not mention indirect rupture of the lamina cribrosa in the *Handbuch der gesamten Augenheilkunde* (von Graefe and Saemisch), nor did von Szily⁶ in his atlas of war injuries. Rönne⁷ considered that "evulsion of the optic nerve" had been described from twenty to twenty-five times but that it must in all probability be much more common. Only five of the instances which he collected from the literature had been caused by indirect injury. Lister,⁸ discussing concussion changes met with in military practice, suggested the name "expulsion of the optic nerve" for the injury which is caused by contusion of the ocular globe.

A report of the microscopic observations in two cases follows. In both instances the eyeball was removed because of painful traumatic glaucoma some time after the injury, a severe contusion of the eyeball. In neither case could the condition of the optic nerve and lamina cribrosa be examined clinically because of hemorrhage into the vitreous.

From the Department of Ophthalmology of the University of Amsterdam (Prof. W. P. C. Zeeman, M.D.).

1. Parsons, J. H.: *The Pathology of the Eye*, London, Hodder & Stoughton, 1908.

2. Lang, W.: Rupture of the Lamina Cribrosa and Optic Nerve-Fibers at the Papilla, *Tr. Ophth. Soc. U. Kingdom* **21**:98, 1901.

3. Cramer, in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1931, vol. 4.

4. Gonin, J.: Ruptures partielles de la papille optique, *Ann. d'ocul.* **147**: 16, 1912.

5. Wagenmann, A.: Die Verletzungen der Auges, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 3, Leipzig, Wilhelm Engelmann, 1915.

6. von Szily, A.: *Atlas der Kreigsaugenheilkunde*, ed. 3, Stuttgart, Ferdinand Enke, 1918.

7. Rönne, H., in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 5.

8. Lister, W.: Some Concussion Changes Met with in Military Practice, *Brit. J. Ophth.* **8**:305, 1924.

In both cases the microscope showed also an atypical, incomplete rupture of the sclera. This type of rupture is new to the literature and will be described in a separate article.⁹ The serious lesions in the retina, choroid, ciliary vessels and nerves will be described in an article on traumatic glaucoma.

REPORT OF MICROSCOPIC OBSERVATIONS IN TWO CASES

CASE 1 (fig. 1).—The optic nerve exhibited a very deep funnel-shaped excavation which ran through the lamina cribrosa to the exterior of the eyeball. As the nerve had been cut rather short in the operation for removal of the eyeball, it was impossible to determine how far the excavation had continued into the nerve.

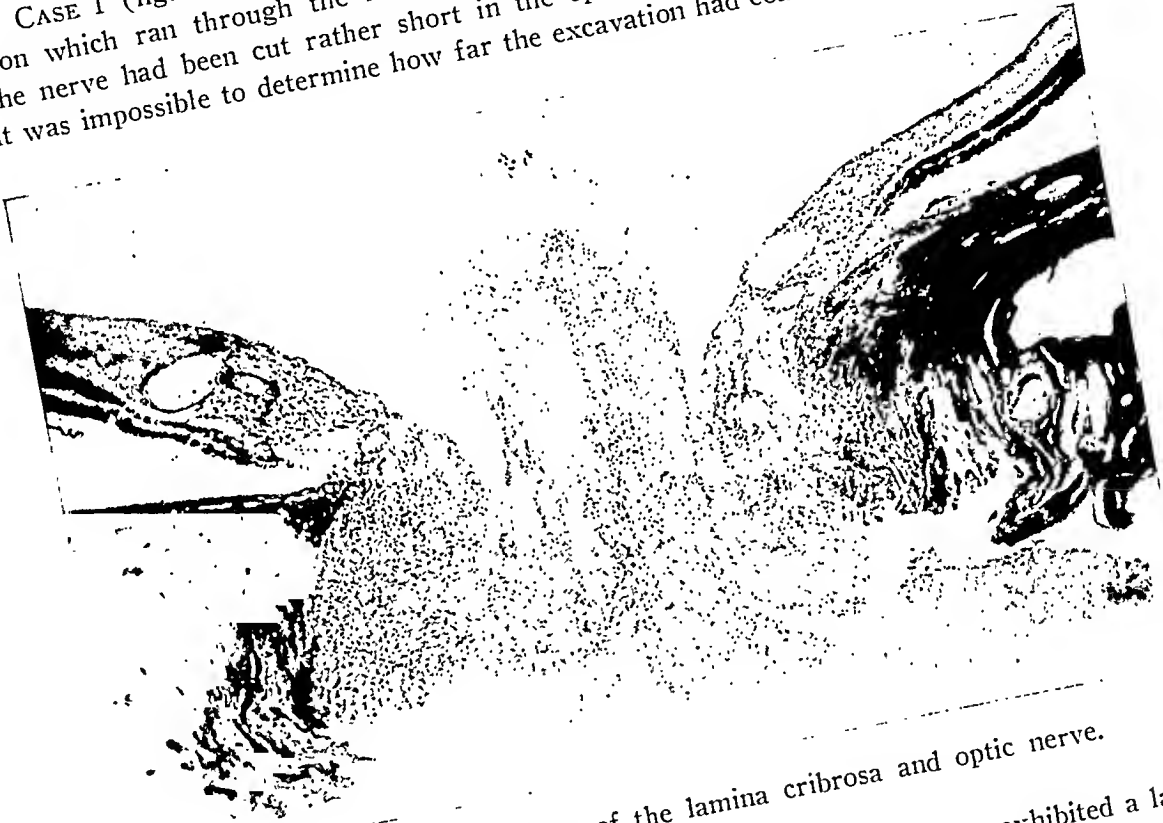


Fig. 1 (case 1).—Rupture of the lamina cribrosa and optic nerve.

The lamina cribrosa had been bent backward and at its center exhibited a large defect through which the optic cup passed outward. This cup differed from a glaucomatous cup by its unusual shape and by the direction of the anterior fibers of the lamina cribrosa. Moreover, glia and nerve tissue were seen anterior to the lamina. The posterior fibers of the lamina had been severed in the operation. The anterior fibers, however, were clearly seen in the microscopic sections and appeared to have been straightened in a posterior direction. Anterior to them the columns of optic nerve fibers gave the impression of having been displaced backward.

CASE 2 (fig. 2).—The optic nerve exhibited a markedly glaucomatous cup and a few caverns. It was also partly atrophic, as was demonstrated by staining the myelin sheaths.

9. Tillema, A.: Atypical, Indirect, Incomplete Rupture of the Sclera. *Brit. J. Ophth.* 20:193, 1936.

Lateral to the central vein an irregularly sausage-shaped cavity was seen which opened anteriorly into the vitreous body and posteriorly into the central vein. It was not lined with endothelium but was filled with a loose tissue derived probably from the vitreous and containing a fair amount of connective tissue. Cells with a large oval pale nucleus and cells with a rather dark oval nucleus (probably fibroblasts) were scattered irregularly throughout this tissue. In a few places young vessels were entering. Some of the cells contained fine pigment granules. This mass of tissue projected posteriorly into the lumen of the central vein.

Medial to this cavity the central vein was found, and medial to the vein, the artery. The latter showed an uninterrupted course from the optic nerve into the retina. The vein, however, was interrupted. Coming from the retina and turning posteriorly along the floor of the glaucomatous optic cup it was still filled with red blood corpuscles. Close to the cavity it made a sharp turn in a posterior

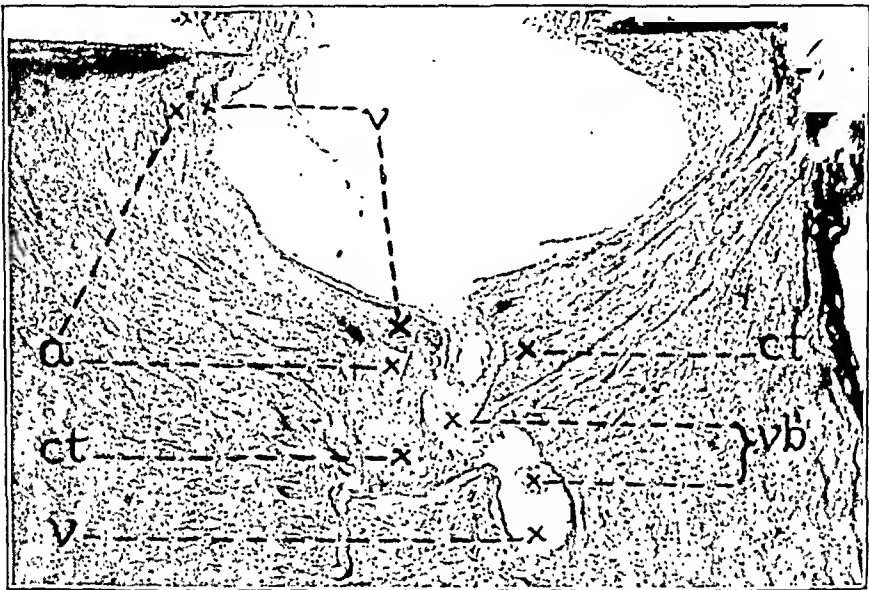


Fig. 2 (case 2).—Rupture of the lamina cribrosa and central vein: *a*, central artery; *ct*, young connective tissue; *v*, central vein; *vb*, vitreous body.

direction and ended as a delicate tube of endothelial cells on the medial wall of the cavity. Farther back behind the lamina cribrosa one picked it up again, containing only a few white blood cells. Much farther back it received many small tributaries and was full once more. It will be noted from this description that the central vein and the cavity appeared side by side anteriorly but that they continued as one structure posteriorly.

In the region of the lamina cribrosa the vein, artery and cavity were surrounded by a mass of fairly dense irregular connective tissue. This tissue was very cellular and contained blood capillaries. Like the tissue filling the cavity, it contained a few pigment granules here and there. From the lamina cribrosa it continued backward, surrounding the central vein and containing a large number of lymphocytes. A little farther back it faded out.

Inasmuch as no junction of veins was found in the optic nerve, the superior and inferior central veins must have joined in the retina.

Continuation of the optic cup backward is found both as a congenital anomaly (Hagedoorn¹⁰) and is a feature of glaucoma (Elschnig¹¹). In both instances the condition vaguely resembles the prolapse of the vitreous described in a foregoing paragraph. The rupture of the central vein, however, and the presence of a mass of young connective tissue containing pigment granules leave no doubt as to the traumatic origin of the condition that has been described.

10. Hagedoorn, Å.: Ein anatomischer Beitrag zur Frage der umschriebenen Grubenbildung am Sehnerveneintritt, *Arch. f. Augenh.* **99**:387, 1928.

11. Elschnig, A.: *Augenärztliche Unterrichtstafeln*, Breslau, J. V. Kern, 1900; in Henke, F., and Lubarsch, O.: *Handbuch der speziellen pathologischen Anatomie und Histologie*, Berlin, Julius Springer, 1928, vol. 11.

HUMAN CYCLOPIA

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The ophthalmologic clinic of the Northwestern University has received from the obstetric department of the Michael Reese Hospital, through the kindness of Dr. Harry Gradle, the head of a cyclopic fetus, concerning which, because of its rarity, I wish to make a report. To show the variation in degree of singleness in these cases, descriptions of two other cyclops from the Anatomic Museum of the Northwestern University Medical School have been included. The first of these (fig. 1) had a single median orbit, four lids forming a single diamond-shaped palpebral fissure in which were seen two bulbs of about normal size for its age, each with its cornea 5 mm. in diameter. The two bulbs fused posteriorly in the midline. Above the orbit was a proboscis 1 by 1.5 cm. No other deformity of the head was apparent. The second cyclops (fig. 2) had a single median orbit similar to the first, containing a single eye slightly larger than normal, with a single horizontally oval cornea measuring 4 by 7 mm. Above this was a proboscis 1 cm. in diameter and 2 cm. long. The third specimen (fig. 3), which is the subject of this report, had a deep horizontal furrow below the proboscis, and deep in the orbit was a microphthalmic eye which was not visible from the outside. (The lower part of the head in figure 3 had been cut off and a cut made through the skin above the proboscis.)

REPORT OF A CASE

The history in the third case was as follows:

Mrs. W., the mother, had one normal child, 5 years of age. During the third, fourth and fifth months of the present pregnancy there had been considerable nausea and vomiting, slight edema and occasional dysuria. The urine showed a trace of albumin, which persisted in spite of a diet poor in salt. The blood chemistry was normal. Physical examination showed a blood pressure of 168 systolic and 96 diastolic and a moderate enlargement of the thyroid gland. At nine months, on Oct. 3, 1930, a fetus weighing 1,440 Gm. was born. It had a single orbit with a proboscis above; the left hand had six fingers, and the genitalia were incompletely developed male organs. In 1934 Mrs. W. delivered a normal healthy child after a normal pregnancy. There have been no other pregnancies.

The head of this fetus was preserved in a solution of formaldehyde and was received at the laboratory with the lower part removed. It measured 9 cm. antero-posteriorly and 7 cm. bitemporally. Across the face was a horizontal furrow

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which showed histologically normal lid structures. Some tissue, later found to be conjunctival tissue, protruded from the center of the furrow. Above this was the proboscis. No other gross deformities were seen. The head was decalcified, embedded in pyroxylin and sectioned from above downward, and every tenth section through the region of the eye was stained with hematoxylin and eosin. The orbit



Fig. 1.—A cyclops from the Anatomic Museum of the Northwestern University Medical School.



Fig. 2.—A cyclops from the Anatomic Museum of the Northwestern University Medical School.

was 25 mm. wide and was filled with fatty areolar tissue in which were two sets of muscles; the small eye was placed superiorly and posteriorly in the orbit. Anteriorly there were a narrow conjunctival sac and lids, and in the center above the lids was the proboscis.

The eye itself consisted of two anterior segments which fused into a single posterior segment from which a single optic nerve ran posteriorly and superiorly into the skull through a bony canal 7 mm. wide. The two anterior segments were unequal in size, the right being 8 mm. in width at its greatest single width and the left 5 mm. in width. The single posterior part was 4 mm. wide. The tunica fibrosa (fig. 4, 1) was a dense fibrous tissue very similar to normal sclera. No differentiation of a cornea could be made out anteriorly, except that the conjunctival sac came in contact with the right half in one place (fig. 4, 2). This layer formed the two anterior segments and fused medially into a single wall, which posteriorly became a thicker mass and extended into each cavity to form a stalk around which the detached retina (fig. 4, 5) and the lens were pressed, leaving little vitreous (fig. 4, 10) but a large cavity anteriorly in the right and a lesser one in the left. In this thicker mass extending into the eye was a sharply

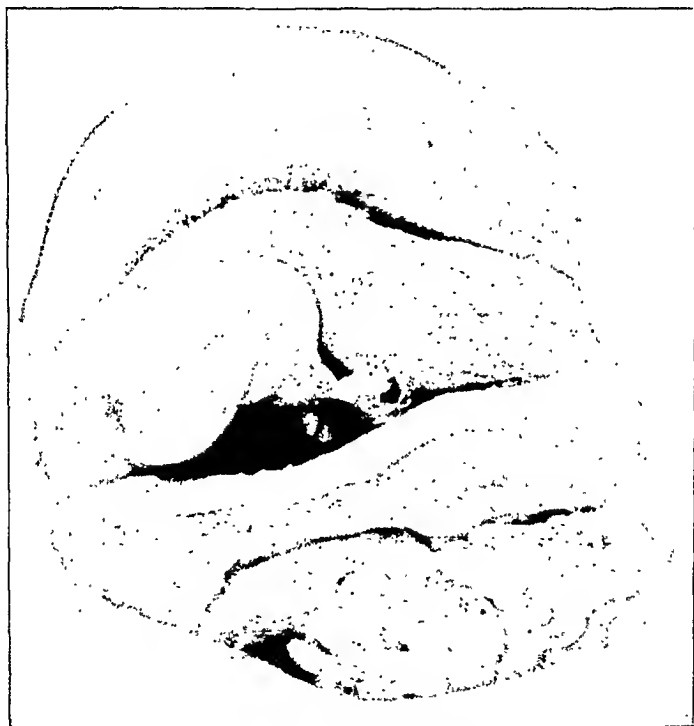


Fig. 3.—The cyclops reported on here.

outlined oval plate of cartilage (fig. 4, 4). In the right cavity the cartilage was 2.5 mm. long, and in the left, about 1.5 mm. The right side contained some vascular granulation tissue (fig. 4, 6) just posterior to the lens. In this area was deposited some calcium. The mass in the right side was adherent to the choroid anteriorly and to the retina on one side (fig. 4, 7). The choroid was a thin layer and contained little blood, only a few medium-sized vessels being visible. Chromatophores were present. Anteriorly, it became difficult to distinguish this from the tunica fibrosa; in one place a thickening was seen which was probably ciliary body and a small bit of iris stroma (fig. 4, 8). A normal layer of pigment (fig. 4, 9) lay in contact with the fibrous layer lining the whole cavity. Where the iris tissue was seen in the right side, a small projection extended posteriorly into the cavity, and from this two fine blood vessels (fig. 4, 11) extended posteriorly to the lens.

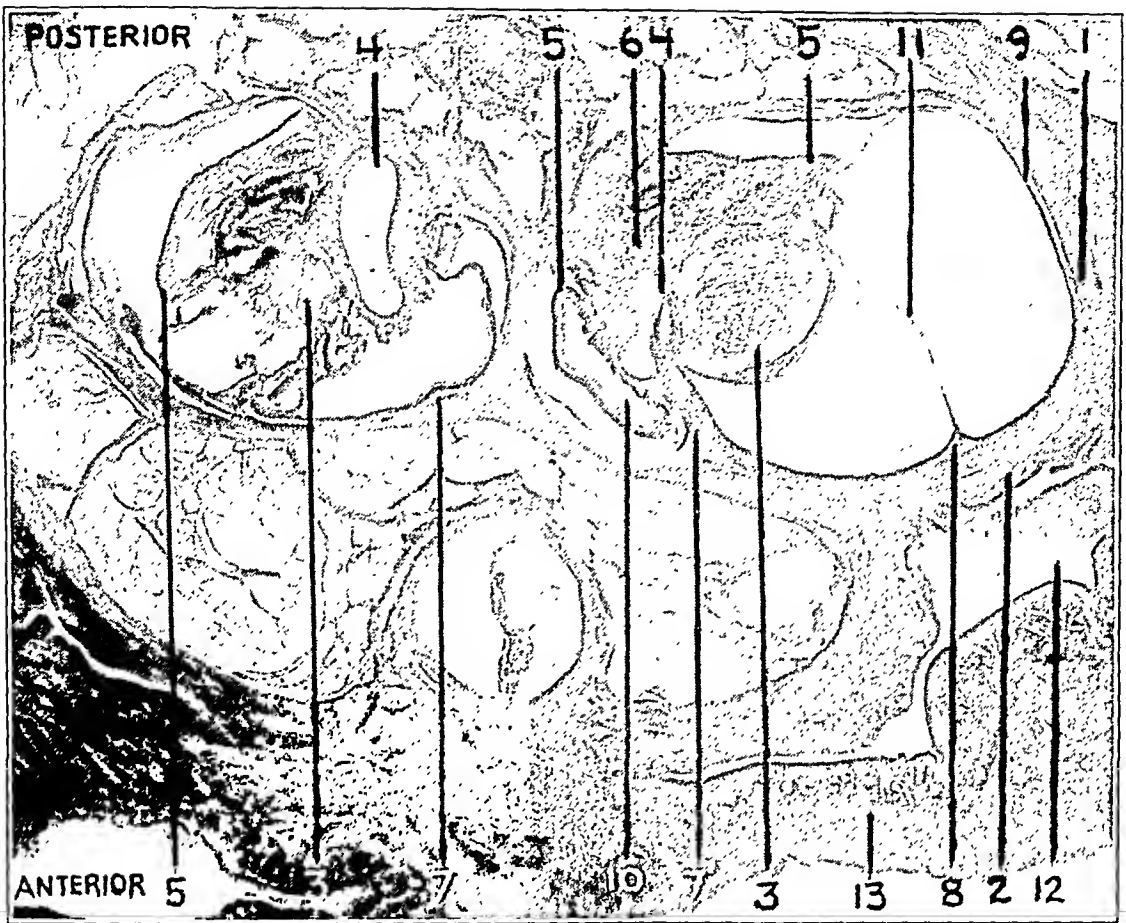


Fig. 4.—Photomicrograph of the eye: 1, the sclera; 2, the cornea; 3, the lens; 4, cartilage; 5, the retina; 6, granulation tissue; 7, the choroid; 8, the iris; 9, pigment; 10, the vitreous; 11, blood vessels; 12, the conjunctival sac, and 13, the lid.



Fig. 5.—Photomicrograph showing the single optic nerve.

The lens (fig. 4, 3) on each side was in contact with the connective tissue mass posterior to it. The right lens was 3 mm. and the left lens 1 mm. in diameter. The capsule and the epithelium of the anterior surface were normal. The substance of the left lens was homogeneous; that of the right was split up irregularly. Anterior to the right lens was a cavity filled with light pink homogeneous fluid. This cavity comprised half of the interior of the bulb. Anterior to the left lens was a smaller cavity. Posterior to the right lens was folded retina (fig. 4, 5) on one side of the lens, and on the other side retina that was smooth (though away from the wall), probably separated during fixation. Within the latter a small amount of vitreous (fig. 4, 10) was present. In the folded part were distinct rosettes. The retina showed two cells layers and in places a ganglion cell layer. The external limiting membrane was present, but there were no rods or cones. In the left half of the eye the retina was not as much folded, but lay more smoothly and more nearly in contact with the walls of the cavity. In several places in the granulation tissue back of the lens and just outside of the sclera were occasional groups of polymorphonuclear cells.

Farther posterior and superior there was a fusion of the two cavities, so that only a single cavity lined with retina was present, and from this the single optic nerve ran posteriorly (fig. 5). This was stained with the Davenport stain for nerve fibers and showed normal young nerve fibers in the optic nerve. No sign of a chiasm was present. The nerve was followed 2 cm. behind the eye but could not be found farther back.

COMMENT

Cases of cyclopia have been known since very early, as indicated by the story of the cyclops in Ulysses. In a textbook on ophthalmology published in 1830, Rosas¹ said: "There are many reports of cyclopic eyes." Twenty-three cases of cyclopia in man were found reported in the literature available. The condition has been reported in horses, birds, rats and fish and is especially frequent in pigs. Although there is great variation in the degree of deformity, the usual picture is partial or complete fusion of the two eyes in the midline with a proboscis above instead of a nose and defects in the forebrain, such as absence of the first cranial nerve and sometimes of the second. Other defects may be present, such as polydactylism, anencephaly and astomatism.

About 1910 there was considerable interest in this condition, especially in the theories as to its cause. These were well presented by Hill.² About the same time Stockard³ reported his experiments on salamander eggs, in which normal eggs produced 50 per cent cyclopic embryos when the magnesium chloride content of the water in which

1. Rosas, cited by Nieden, A.: Ueber Anophthalmia cyclopica, Arch. f. Augenh. **22**:61, 1890-1891.

2. Hill, E.: Cyclopia, Its Bearing upon Certain Problems of Teratogenesis and of Normal Embryology, Arch. Ophth. **49**:597, 1920.

3. Stockard, C. R.: The Artificial Production of One-Eyed Monsters and Other Defects, Which Occur in Nature, by the Use of Chemicals, Anat. Rec. **3**:167, 1909.

they were developing was increased. Stockard expressed the belief that the magnesium had an inhibitory or anesthetic effect on the out-pushing and separation of the optic vesicles. He stated that the eye primordium is in the midventral line of the brain and that early arrest of development causes this to remain median or divide in part to form the hour-glass-shaped eye. Wolff ⁴ has produced cyclopia in chicks by irradiation at an early stage.

An interesting point is the frequent finding of a single posterior part and a double anterior segment of the eye. In twelve of sixteen cases in which the interior of the eye was described, there were two lenses but a single optic nerve. It seems probable that the single optic vesicle stimulated the surface ectoderm on each side of the head, causing the formation of two lenses which conditioned the formation of the other structures of the anterior segment about the lenses, making an apparent two eyes from the initial single vesicle. This would fit in well with the theory that the eyes come from a single median anlage which separates normally into two, but that in cyclopic eyes this separation fails to take place, rather than with the theory that there are originally two separate anlagen which in cyclopic monsters become fused. This has been a point of dispute.

Another interesting finding is the rosettes of the retina. This was mentioned by Hill ² in his case report, but he found by staining with a special stain for neuroglia that there was only a superficial resemblance. Rosettes are due to excessive growth of the primitive outer neuroblastic layer which furnishes nuclei of cones and rods and becomes the outer nuclear layer. Goldstein and Wexler ⁵ found such rosettes in embryos in cases in which roentgen irradiation had been used to terminate pregnancy during the second month.

The finding of cartilage in the eye in cyclopean eyes has been mentioned in the literature. Certain reptiles have plates of cartilage in the eye. The significance of this in the case reported here is not clear. The cartilage may be the beginning of the bone formation that occurs in shrunken or degenerated eyes. The granulation tissue and the few polymorphonuclears indicate that the early injury which this embryo sustained was probably an infection rather than a chemical or physical injury.

122 South Michigan Avenue.

4. Wolff, E.: Recherches expérimentales sur la cyclopie, *Arch. d'anat. d'histol. et d'embryol.* **18**:145, 1934.

5. Goldstein, I., and Wexler, D.: Rosette Formation in Eyes of Irradiated Human Embryos, *Tr. Am. Acad. Ophth.* **35**:140, 1930.

MELANOCARCINOMA OF THE CONJUNCTIVA

REPORT OF A CASE

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AND

MAURICE P. S. SPEARMAN, M.D.

EL PASO, TEXAS

Epithelioma of the conjunctiva may vary in malignancy, but in about 80 per cent of the cases it tends to recur, even when superficial. If the diagnosis is made early, it is proper to remove the tumor; but if the eyeball itself is involved, complete enucleation of the globe, together with exenteration of the remainder of the orbital contents, is indicated. Some form of radiation therapy should be employed after following surgical intervention.

Malignant growths in the bulbar conjunctiva are often the result of extension from a primary focus in the conjunctiva of the lid. This primary focus may be a nevus, which is a congenital growth usually considered benign. It is a smooth, flat tumor highly pigmented with melanin. The color may vary from light to dark red-brown. These nevi of the conjunctiva have much the same structure histologically as that of the ordinary pigmented nevi found on the skin elsewhere on the body. Nests of pigment-bearing cells lie in a network of connective tissue, with conjunctival epithelium penetrating into the structure. Several theories as to the origin of these nevus cells have been advanced. We may mention here: (1) the theory that they derive from the endothelial cells of the lymph vessels, as was advocated by Recklinghausen; (2) the view that they arise from the surface epithelium, as was set forth by Unna,¹ and (3) Ribbert's contention that they come from the chromatophores, which are situated in the delicate connective tissue of the conjunctiva. Bloch,² in a brilliant piece of chemical research, developed the so-called dopa reaction, wherein he showed that the epidermal cells alone produce melanin in normal tissues. D'Agata³ later showed that the only cases in which connective tissue or endothelial cells could be shown to be melanin-producing by the dopa reaction were those of tumor. It is felt by most workers today that pigmented nevus cells arise from the epithelium. Hence pigmented tumors that are

1. Unna, P. G.: Berl. klin. Wchnschr. **30**:14, 1893.

2. Bloch, B.: Ztschr. f. phys. Chem. **98**:226, 1916-1917.

3. D'Agata, G.: Tumori **9**:121, 1922.

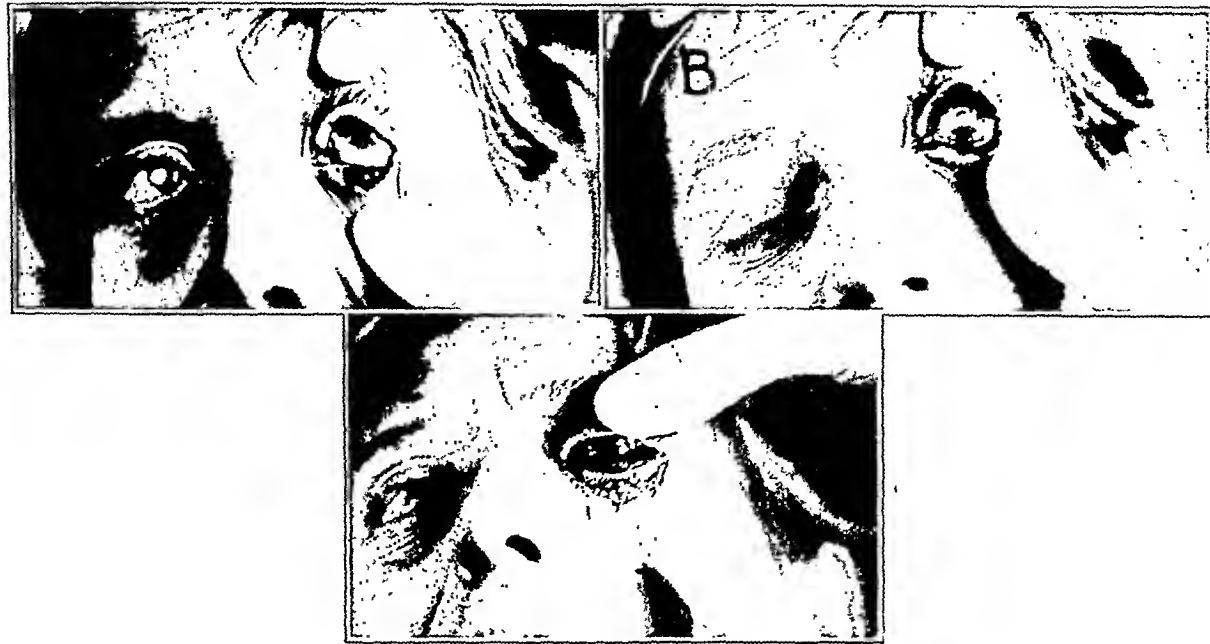


Fig. 1.—Photograph of the patient. *A* shows invasion of conjunctiva of the left lower lid by melanocarcinoma; *B*, involvement of the bulbar conjunctiva, and *C*, involvement of the conjunctiva, with the left upper lid everted.

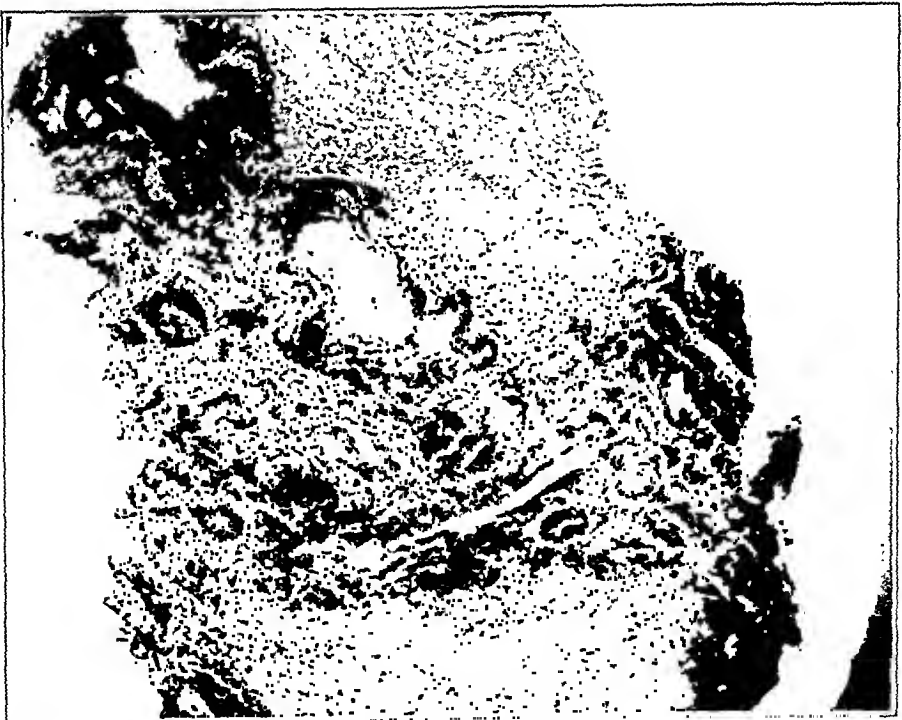


Fig. 2.—Microscopic section of the melanocarcinoma; low power magnification.

derived from pigmented nevi would more properly be called melanocarcinomas rather than sarcomas.

Melanocarcinoma shows no marked tendency to penetrate the eyeball in its early stages. Later it may grow over the cornea, thus impairing vision. The entire conjunctival sac may be invaded. Frequent, alarming hemorrhages often occur. Metastases sooner or later end the patient's life.

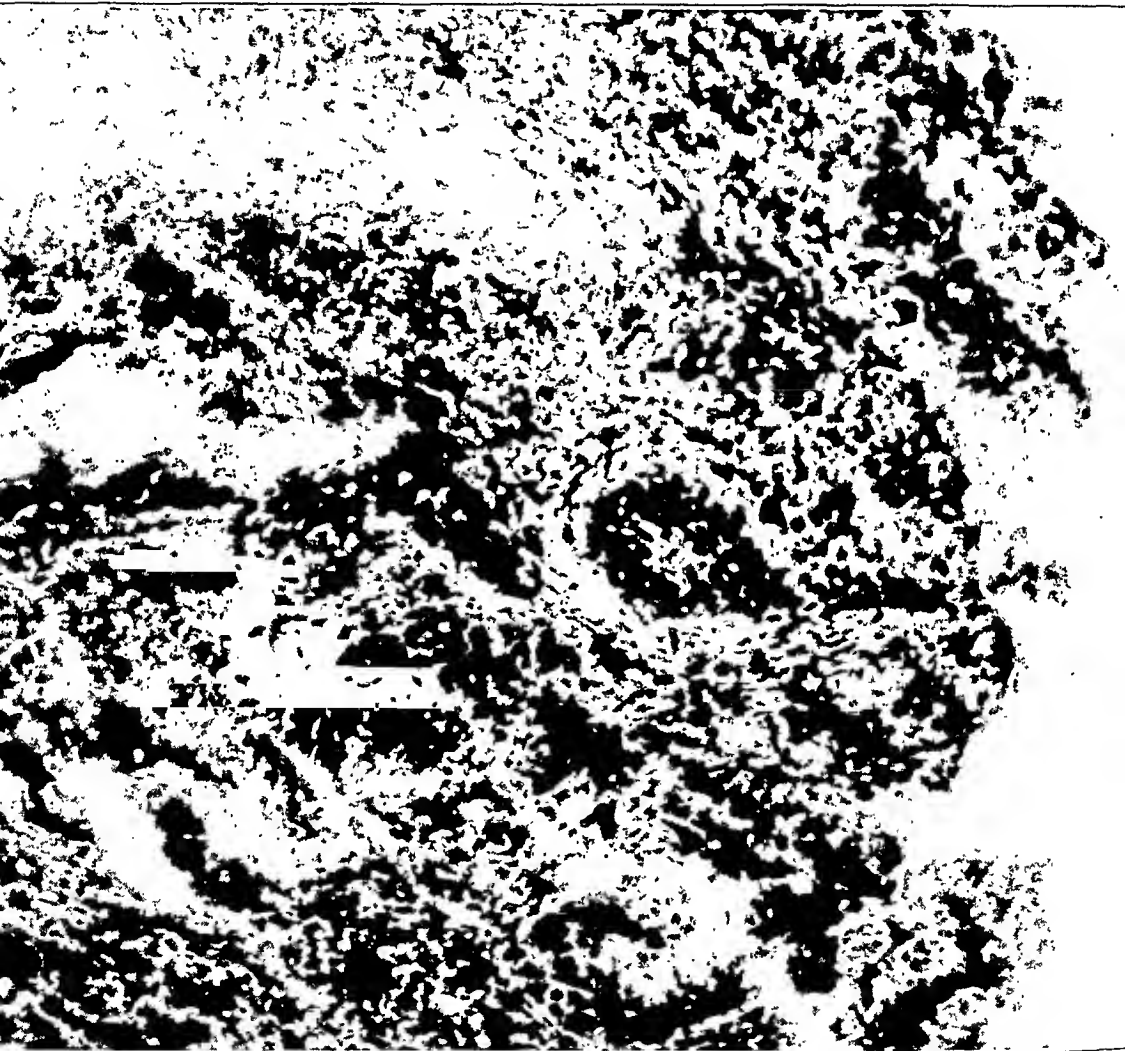


Fig. 3.—Microscopic section of the melanocarcinoma; high power magnification.

The diagnosis of melanocarcinoma is made from (*a*) the history, (*b*) the clinical examination, (*c*) biopsy of sections and of pathologic tissue and (*d*) the dopa reaction.

We herewith report a case of melanocarcinoma involving the greater portion of the palpebral conjunctiva of the left eye and extending to the bulbar conjunctiva. This malignant growth seemingly arose from

a pigmented nevus on the internal conjunctival surface of the upper lid of the involved eye.

REPORT OF CASE

I. E., a Mexican widow, aged 50, presented herself for consultation on Oct. 22, 1935. Her complaints were: (1) itching of the left eyeball and lids, (2) excessive watering of the left eye, (3) a slight bloody discharge from the left eye and (4) a small dark brown mass projecting from the under part of the left upper lid.

Six months previously the patient began to notice an increasing itching sensation of the left eye and lids. There was an unusually copious flow of tears. While facing a mirror one day she noticed a small dark brown spot on the left upper lid, behind the lashes. She turned the lid up slightly and noticed a dark substance covering a portion of the inner surface of this lid. Treatment consisting of frequent irrigations of the eye with water was instituted in the home. About one month later she first noticed that the tears were often slightly blood-tinged. As time passed she found that her pillows were blood-stained when she awoke in the morning.



Fig. 4.—Photograph of the patient taken on Jan. 21, 1936, showing definite progress of the tumor and enlargement of the anterior preauricular glands.

Examination on October 22 revealed a rather emaciated, apprehensive middle-aged woman. The right eye presented an old leukoma of the cornea that completely covered the pupil. Vision in this eye was but questionable perception of light. An internal squint was present. Examination of the left eye showed vision of 1.2. In this eye there were no squint, no abnormal position of the lids, no ulceration of the margins of the lids and no inflammation of the lacrimal apparatus. The cornea was clear. The palpebral conjunctiva of both lids exhibited several isolated patches of a dark brown, friable substance. Hemorrhage followed attempts to dislodge this material. One patch on the upper lid was the largest, being about 0.5 cm. in diameter. The temporal aspect of the bulbar conjunctiva exhibited, near the superior fornix, several round areas of the same dark brown substance, none of which was larger than 0.1 cm. in diameter. The intra-ocular tension of the eye was not increased. The pupil was regular and active to light and in accommodation. The media were clear. The fundus showed no pathologic changes.

A pathologist was called in conference. He examined a section of the suspected material and gave an opinion that the condition was melanocarcinoma. A roentgenologist was consulted. He felt that the condition was too far advanced to be helped by radiation therapy.

Mild astringent eye drops were prescribed, and the patient was requested to return at intervals.

On November 6 the patient was seen by us for the second time. The growth had progressed little. She was experiencing no pain. On November 15 she complained of intermittent sharp pains in the left eyeball, frequent severe frontal headaches, transient dimness of vision and a burning sensation in the left eyelids and the nose. At this time the growth under the left upper lid had begun to pile up in such fashion that the lid was being markedly displaced outward. The areas of the growth that were once discrete had become coalesced. Little of the palpebral conjunctiva remained uninvolved. The nasal half of the bulbar conjunctiva and the cornea were clear. The patient mentioned during this visit that she had had several mild hemorrhages from the left side of the nose. Examination of the nose revealed no gross signs of metastasis, however.

Since (*a*) this patient was totally blind in the right eye, (*b*) the disease process of the left eye was too far advanced for radiation therapy to be of help, (*c*) total exenteration of the left orbit was probably the only choice remaining and (*d*) there was a possibility that metastases had already progressed by extension down the left nasolacrimal duct, we were compelled to adopt a highly unsatisfactory program of treatment, that is, treatment solely palliative in intent.

SUMMARY

A case is reported of melanocarcinoma of the palpebral and bulbar conjunctiva with possible metastasis by extension down the nasolacrimal duct.

FILTRATION EXPERIMENTS WITH THE VIRUS OF INCLUSION BLENNORRHEA

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AND

SANFORD R. GIFFORD, M.D.

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The evidence for the etiologic significance of certain morphologic elements (elementary and initial bodies) observed constantly in the cytoplasm of the epithelial cells of the conjunctiva in inclusion blennorrhea of the new-born has recently been strengthened by the experiments of Thygeson,¹ who reported his investigation in eleven cases of the disease. His statement that these inclusions are the causal agents of inclusion conjunctivitis is based on (1) their constant presence, (2) their absence in bacterial conjunctivitis, (3) the absence of pathogenic bacteria in inclusion blennorrhea, (4) the transmissibility of inclusion conjunctivitis and the presence of inclusions in the new host and (5) the production of the disease with bacteria-free suspensions of epithelial scrapings containing inclusions.

Two patients with inclusion blennorrhea were available to us for detailed study and inoculation of animals, and the positive results obtained seem to justify their being reported here. The graded collodion membranes² used in our experiments were prepared by the method described in detail by Bauer and Hughes.³ The material for filtration was prepared as described by Thygeson, and the smears stained with Giemsa's solution were decolorized slightly, as he suggested, in order to facilitate the differentiation of the inclusion bodies from leukocytic granules. The filtrations were carried out under a 50 pound (22.7 Kg.) pressure (nitrogen) in the Bauer and Hughes filter chamber.⁴ The suspensions were first freed from particles of tissue by centrifugation; hence, the clogging of the membrane was reduced to the minimum, and nearly the whole of the fluid was recovered in the filtrate. The protocols for the two experiments follow.

From the Departments of Research Bacteriology and Ophthalmology, the Northwestern University Medical School.

1. Thygeson, P.: *Am. J. Ophth.* **17**:1019, 1934
2. Elford, W. J.: *J. Path. & Bact.* **34**:505, 1931.
3. Bauer, J. H., and Hughes, T. P.: *J. Gen. Physiol.* **18**:143, 1934.
4. Dr. Bauer had one of these filter chambers made for us.

EXPERIMENTAL INVESTIGATION

EXPERIMENT 1.—In A. S. conjunctivitis developed thirteen days after birth. Examination of smears and scrapings failed to show gonococci or other bacteria but revealed many typical inclusions. Material for experimentation was taken thirty-one days after birth by gentle removal with a platinum spatula of epithelium from the conjunctiva of the upper and lower lids of both eyes. The material was suspended in 2 cc. of plain infusion broth; the shreds were broken by grinding in a mortar, and the tissue débris was removed by centrifugation for ten minutes at low speed. Six drops of the clear supernatant fluid was instilled into the right eye of baboon 1 (sphinx baboon, *Papio sphinx*) after preliminary scarification of the conjunctiva of the upper and lower lids. The remainder of the fluid was filtered through a graded collodion membrane with an average pore diameter of 0.46 micron, and was then repeatedly instilled into the scarified conjunctiva of the right eye of baboon 1F, all the filtrate being utilized in this way. The monkeys were separately caged throughout the experiments. The unfiltered material yielded a few colonies of *Staphylococcus albus* and diphtheroid bacilli on culture on plain agar and broth, blood agar and semisolid 10 per cent serum (*Leptospira*) medium. The filtrate was bacteriologically sterile, and the membrane failed to permit passage of a culture of *Bacillus prodigiosus* as indicated by tests after filtration of the experimental material.

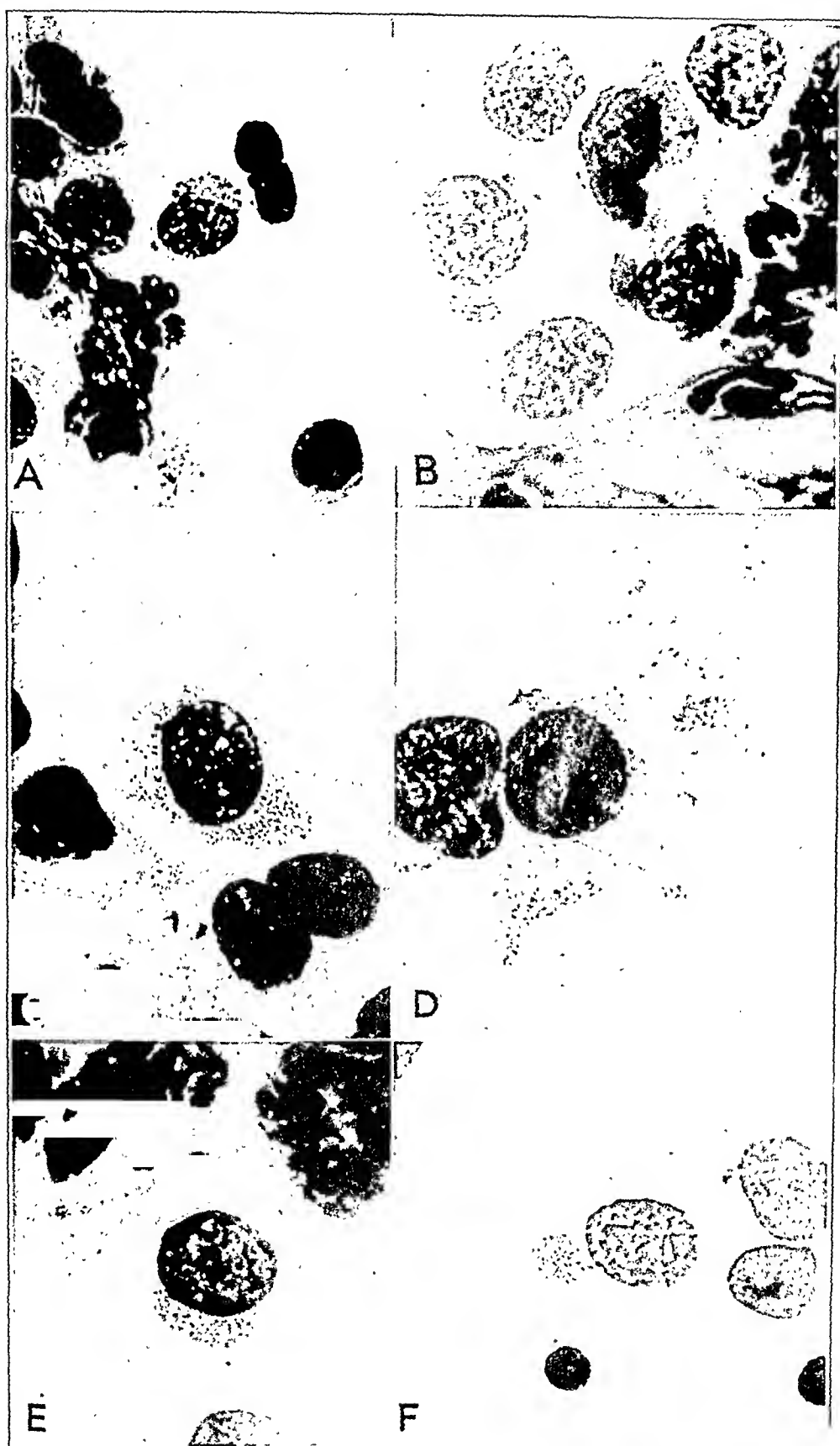
After forty-eight hours, in baboon 1 moderate swelling and redness of both the upper and the lower fold were evident, and in baboon 1F there was slight swelling of the lower fold.

After ninety-six hours, swelling and redness were present in both folds of the inoculated conjunctiva of both animals. The upper tarsus was not affected in either animal.

After one week, the tarsus and the upper fold were clear in both monkeys, and the lower fold showed the same condition as before, the swelling being considerably greater in baboon 1 and the semilunar fold being involved. In both animals there was considerable secretion. The left eye, which had not been inoculated, was not affected in either animal.

After twelve days, diffuse conjunctivitis resembling human inclusion blennorrhea, with involvement of the semilunar fold, was shown on the side of inoculation in both animals. Smears stained with the Giemsa solution showed a number of typical inclusions in the epithelial cells from the eye of baboon 1 (figs. *A* and *B*). No inclusions were observed in the scrapings taken from baboon 1F, and a week later, i. e., at the end of twenty-one days, this animal was definitely recovering, while the condition of baboon 1 remained the same and inclusions were still present in the smears. Twenty-four and thirty-three days after inoculation smears were again made of material obtained from each animal, but no inclusions were noted. Baboon 1F had completely recovered on the twenty-fourth day, but baboon 1 continued to show considerable redness, swelling and mucous secretion.

EXPERIMENT 2.—In each animal the eye which had not been inoculated (the left) remained free from infection. This eye was inoculated at the end of forty days with epithelial scrapings taken from baby W., smears of which (made three days prior to inoculation of the monkey and ten days after birth) contained numerous mixed inclusions (elementary and initial bodies) but no gonococci or other bacteria (figs. *C* and *D*). The preparation of the material and the amounts used were the same in this experiment as in experiment 1, and the method of inoculation was similar, except that 0.2 cc. of the material was injected subconjunctivally and the lids were scarified with the charged needle. The remaining



Photomicrographs of portions of smears prepared from epithelial scrapings. Smears were fixed for fifteen minutes in methyl alcohol, stained two hours in Giemsa's solution (1 drop of the stain to 10 drops of distilled water), decolorized with two changes of 95 per cent alcohol for five seconds each, washed in water, dried in the air and mounted in cedar oil.

A ($\times 750$) shows a mixed inclusion in material obtained from baboon 1 in experiment 1; *B* ($\times 1,175$), several cells from the same monkey, each containing more than one inclusion; *C* ($\times 1,175$), elementary body inclusion, from baby W; *D* ($\times 1,175$), a ruptured cell, with scattering of the elementary bodies, from baby W; *E* ($\times 1,175$), mixed inclusion, from baboon 1F in experiment 2, and *F* ($\times 1,050$), elementary body inclusion, from baboon 1F in experiment 2.

fluid was repeatedly instilled after scarification. Baboon 1F was used for the filtrate, as before, and baboon 1, for the unfiltered material. The average pore diameter of the membrane used in this experiment was somewhat larger (0.62 micron) than that of the membrane employed in experiment 1, but *B. prodigiosus* failed to pass through the filter, and the filtrate was bacteriologically sterile.

After six days, slight swelling and redness were present in both monkeys.

After sixteen days, definite lesions in both the upper and the lower lid of the left eye were evident in both animals, the tarsus also being slightly reddened. Smears, however, showed no inclusions at this time.

After twenty-five days, severe inflammation was present in both animals, and smears showed typical inclusions in both instances (figs. *E* and *F*).

The condition began to regress about this time, and inclusions were not observed on subsequent examination, though follicles were present and the swelling persisted for four months in both animals.

SUMMARY

The two experiments reported in this paper furnish additional evidence that the virus of inclusion blennorrhea is filtrable through graded colloidal membranes with an average pore diameter of 0.46 to 0.62 micron and is transmissible to the sphinx baboon. The morphologic elements (elementary and initial bodies) observed constantly in the cytoplasm of epithelial cells of the conjunctivae of babies with the acute stage of inclusion conjunctivitis were observed to be associated with the disease in the baboon, whether produced by filtered or by unfiltered material. Although inclusions have been observed in experimental inclusion blennorrhea produced by filtrates in man (Thygeson¹), they have not been reported in the disease produced by filtrates in baboons. The observation of inclusions in material obtained from baboons reported in this paper add further evidence of their significance in the etiology of the disease.

EPITHELIAL INLAY IN CASES OF REFRACTORY ECTROPION

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MONACO, MONACO

Nearly twenty years ago I introduced into plastic surgery a method to transplant free skin flaps of varying size. This method is now generally accepted in reconstructive surgery under the name of epithelial inlay and is highly esteemed not only in Austria and Germany but also in England and in the United States.¹

This method may also be used in ophthalmic practice when after enucleation the conjunctival socket is not large enough to receive a glass eye, when there is extensive mutilation of the eyelids by fire, when it is necessary to construct new eyelids, etc. I shall now consider its value only in cases of ectropion in which the usual operations may have been unsuccessful. The ectropion may be the result of a burn, lupus or other accident or illness.

The principles of the epithelial inlay are that an exact model is made with Stent's mass, a material used by dentists to get the shape of the jaw. The material becomes soft when placed in hot water and then becomes stiff in a few minutes if placed on the jaws, or, in the case of epithelial inlay, on wounds, where free skin grafts are to be used. The areas may be of any size, as plenty of thin skin is available on both thighs; in case more skin is needed than can be removed from the thighs, it can be taken from anywhere on the body. I have constructed new urinary bladders and esophagi by this method. The model taken of the wound is covered with a skin graft. The thinner the graft, the surer it will heal. The outer side of the skin graft is placed on the model which is replaced on the wound for one week exactly as it was when it was made. The skin is taken from the inner and anterior side of the thigh, or, if only small pieces are wanted, it may be taken from the inner side of the upper portion of the arm, as the skin is very thin in both places. Firm pressure on the wound prevents the accumulation of any secretion between the wound and the graft. With other methods the secretion would separate the graft from the wound entirely or partly. If the technic of the epithelial inlay is carried out correctly, the graft heals completely, and though I have made many hundred epithelial inlays, I have considered the result unusual if the graft had failed to "take" when the Stent mass was removed after a week. The wounds of

1. Reconstructive Surgery, book review, Brit. M. J. 1:203 (Feb. 2) 1935.

the thigh heal in a few days under a dry dressing or one treated with petrolatum, and afterward, if necessary, other grafts can be taken from the same spot if the grafts are thin. The dressings are left on the thigh until they become displaced of themselves.

REPORT OF A CASE

The following case illustrates my procedure:

A Spanish girl, aged 17, fell into a fire during an epileptic attack and was seriously burned on the face. After the burns healed large scars disfigured the

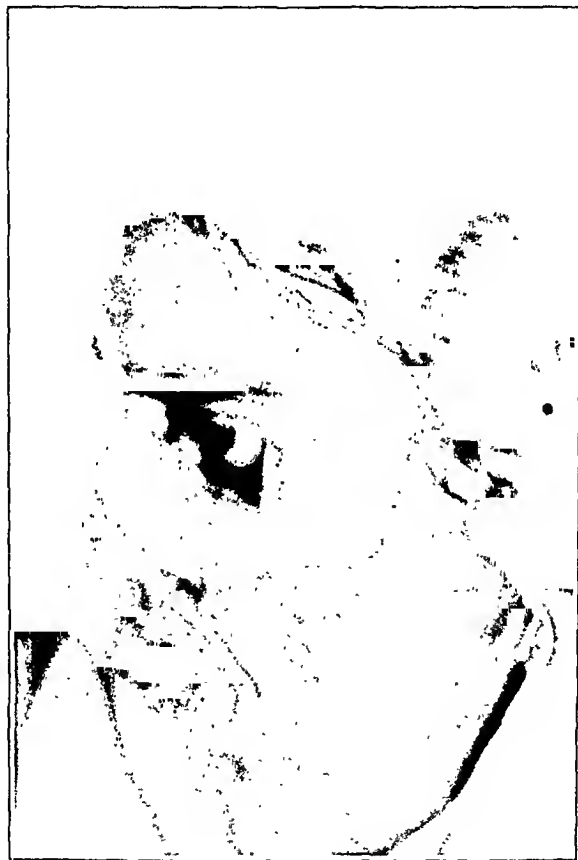


Fig. 1.—Photograph of patient before grafting of epithelial inlays.

face and neck, and the patient could not lift her head (fig. 1) after the accident. The absence of a lower lip and left lower eyelid caused much annoyance and depressed the patient greatly, and the latter constituted a danger to the eye. In addition there was an ectropion of the right lower lid. A surgeon had tried in vain to make a new left lower lid by using two large pedicled skin flaps, one from the forehead above the right eyebrow and one from the cheek in front of the left ear. These flaps were sewed together, but the result was nil, and the disfigurement was much increased. The right eyebrow was displaced nearly 2 cm. above the left, and there were disfiguring scars on the forehead and especially on the cheek. The ectropion was maximal, and there was practically no lower eyelid.

Professor Marquez, the president of the faculty of medicine of the University of Madrid, sent this patient to me, and I operated on her on March 8, 1935, in the surgical clinic of Professor Estella, University of Madrid.

I made four epithelial inlays at one time: one large one for the neck (a whole thickness graft), another to make a new lower lip and one for each ectropion. All four healed.

I shall not speak here of the first two but shall limit my remarks to the last two, as they constituted ophthalmologic problems. First of all, I made a deep cut along the eyelid, nearly at right angles to the surface and somewhat inclined, in order to separate the deep lying conjunctiva and the muscles close to it, which were not cicatricial, from the greater part of the muscles of the lid, which grew more and more scarlike the nearer they approached the skin of the cheek. Then I performed a tarsorrhaphy on each eye and fastened the sutures to the skin above



Fig. 2.—Photographs of patient three weeks after grafting of epithelial inlays.

the eyebrows in order to raise the palpebral fissures as much as possible and to spread the margins of the wound.

Models in Stent's mass were made of both large wounds, and skin grafts from the thigh were placed on the part of each model which was to come in contact with the wound. Then the molds were placed on the wounds, and sutures of strong silk were applied tightly in U shape over the molds from the forehead to the cheek to press the molds against the wounded surface. After a week the sutures were removed. The results three weeks later are shown in in figure 2. At a later date I shall undertake further reparative procedures.

BIOCHEMISTRY OF THE LENS

VII. SOME STUDIES ON VITAMIN C AND THE LENS

JOHN BELLOWS, M.D.*

CHICAGO

The lens, being devoid of blood vessels in postnatal life, is dependent on its surrounding media for nutrition and respiration. However, because of its internal oxidative mechanism, it is partly independent of the aqueous for its respiration. It is fortunate that this is so, for, according to Duke-Elder,¹ the oxygen tension of the aqueous is insufficient to meet the requirements of the lens. The internal oxidative system of the lens functions through the agencies of glutathione and vitamin C, or cevitamic acid. The latter is present in the aqueous and lens, while the former is found only in the lens.

These substances are capable of being reversibly oxidized and reduced, thus forming a so-called "redox" system. This property depends on the ability of glutathione and vitamin C to accept hydrogen, and in this process the hydrogen donor (the lens) is oxidized. That the reversibly oxidized vitamin C is reduced by the lens appears probable from the report of Buschke and Goldmann.² These writers found that vitamin C appears in the reduced form only in the normal aqueous, whereas in the aphakic eye the vitamin C content of the aqueous drops markedly, and that which is present is in the reversibly oxidized form. Vitamin C, like glutathione, is found to be diminished in quantity or absent in the cataractous lens (von Euler and Martius).³

The question whether the absence of cevitamic acid in the cataractous lens precedes the opacification or is merely a result of this condition cannot be answered definitely at present. Some experiments, however, point to the former possibility. Monjukowa and Fradkin⁴ were able to produce cataract in a small percentage of guinea-pigs by feeding them a scorbutic diet, and if the aqueous was drained by paracentesis cataract

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1. Duke-Elder, W. Stewart: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1934, vol. 1, p. 479.

2. Goldmann, H., and Buschke, W.: *Klin. Wchnschr.* **14**:239, 1935.

3. von Euler, Hans, and Martius, Carl: *Ztschr. f. physiol. Chem.* **222**:65, 1933.

4. Monjukowa, N. K., and Fradkin, M. J.: *Arch. f. Ophth.* **133**:328, 1935.

developed in all of the animals. Bietti and Carteni⁵ found that the quantity of cevitamic acid in the crystalline lens fell to a mere trace in guinea-pigs on a diet deficient in vitamin C. I⁶ found that there was a tendency to a low vitamin C level in the blood of cataractous subjects. These facts point to the possibility that a diminution in the vitamin C content of the eye, whether due to a deficiency in the diet or to some barrier in the blood vessels, may result in a cataract. In view of the importance of vitamin C in the physiology of the eye, it was thought that some further experiments with this substance were justified.

METHOD OF DETERMINATION

The method used for the determination of cevitamic acid and glutathione in the ocular tissues consisted of a combination of several procedures. A 2 per cent solution of meta-phosphoric acid was used in the method recommended by Fujita and Iwatake⁷ for the extraction of cevitamic acid and glutathione. The total iodine-reducing substances present were determined by the method of Woodward and Fry,⁸ a procedure which they used for the determination of the glutathione in the blood. The amount of cevitamic acid was estimated by the method of Birch, Harris and Ray⁹ in which sodium 2,6 di-chlorobenzenone-indophenol is used as an indicator. One obtains the quantity of glutathione by subtracting the amount of iodine reduced by the cevitamic acid from the total iodine-reducing substances present.

PROCEDURE

A lens was ground up in a mortar with a fresh 2.5 per cent solution of meta-phosphoric acid (10 cc.) and a little sand. Meta-phosphoric acid was used instead of sulfosalicylic acid because the latter assumes a pinkish color (probably due to iron present in the distilled water) which interferes in determining the end-point for vitamin C. The extraction was carried out twice. The combined liquid was centrifugated. An aliquot portion of the supernatant fluid was placed in a flask; to this were added 2 cc. of a fresh 5 per cent solution of potassium iodide, 2 cc. of a 5 per cent solution of meta-phosphoric acid and 1 drop of a 1 per cent solution of starch. This was titrated against thousandth-normal potassium iodate made up in a 2 per cent solution of sulfosalicylic acid to maintain the desired acidity. The solution was standardized against glutathione, which has a melting point of 190 C. It was found that 3.145 cc. of thousandth-normal potassium iodate was necessary for 1 mg. of pure glutathione. The amount of potassium iodate obtained thus represented the amount of reducing substances present (glutathione and vitamin C).

An aliquot portion of the supernatant fluid was titrated against the standardized dye (sodium 2,6 di-chlorobenzenone-indophenol). The value obtained is the

5. Bietti, G., and Carteni, A.: *Boll. d. Soc. ital. di biol. sper.* **9**:983, 1934; *abstr., Nutrition Abstr. & Rev.* **5**:376, 1935.

6. Bellows, J.: *Biochemistry of the Lens: V. Cevitamic Content of the Blood and Urine of Subjects with Senile Cataract*, *Arch. Ophth.* **15**:78 (Jan.) 1936.

7. Fujita, A., and Iwatake, D.: *Biochem. Ztschr.* **277**:293, 1935.

8. Woodward, G. E., and Fry, E. G.: *J. Biol. Chem.* **97**:465, 1932.

9. Birch, T. W.; Harris, L. J., and Ray, S. N.: *Biochem. J.* **27**:590, 1933.

amount of vitamin C present. By changing this value into terms of thousandth-normal potassium iodate and subtracting this amount from the total quantity of potassium iodate titrated, one obtains the amount reduced by glutathione.

That the method used for the determination was accurate was shown by tests for recovery (table 1). In these tests known amounts of cevitanic acid and glutathione were added to lens extract which had previously been examined for these substances. The amount of recovered vitamin C was 100 per cent, and that of glutathione, 95 per cent.

EXPERIMENT 1.—The glutathione and vitamin C contents of a mature and an immature senile cataract from a human being were determined. The mature senile cataract, weighing 117 mg., did not contain an appreciable amount of glutathione or vitamin C, and the immature senile cataract (brown), weighing 228 mg., contained 81.9 mg. of glutathione per hundred grams and 5.7 mg. of cevitanic acid. According to the figures of Müller and Buschke, the normal lens of a human being contains 29.6 mg. of cevitanic acid per hundred grams and a bovine lens contains 129.7 mg. of glutathione and 32.7 mg. of cevitanic acid.

The figures given here are in accordance with those of other workers. The amounts of glutathione and vitamin C were diminished in the immature cataract, while in the mature cataract these substances were not present.

TABLE 1.—*Results of Tests for the Recovery of Glutathione and Cevitanic Acid*

	Amount Present In Lens Extract, Mg.	Amount Added, Mg.	Amount Recov- ered, Mg.
Glutathione.....	0.081	0.020	0.100 (95%)
Vitamin C.....	0.005	0.012	0.017 (100%)

EXPERIMENT 2.—The absorption of cevitanic acid from the conjunctival sac of a rabbit was demonstrated. Powdered vitamin C¹⁰ was placed in the conjunctival sac of a rabbit, and the closed lids were massaged over this. (A total of 100 mg. was used.) Twenty hours later this procedure was repeated with 50 mg. of the powder. After one hour the conjunctival sac was washed with a solution of boric acid. Following this, under butyn anesthesia, a fine needle at the end of a syringe was introduced into the anterior chamber, and a few drops of aqueous humor was removed. The chamber was not completely emptied, because it was thought that the last of the aqueous would contain some second aqueous, which has been shown to contain little vitamin C. The aqueous of the fellow eye was used as a control, because it was shown by Müller and Buschke¹¹ that the concentration of this substance in either eye of an animal is the same. The aqueous was immediately mixed with a 2 per cent solution of meta-phosphoric acid (fresh) and centrifugated. Aliquot portions of the supernatant fluid were titrated for vitamin C concentration by means of the dye in a microburet graduated in 0.002 cc. The concentration in the treated eye was greater than in the control (table 2).

The values are probably low because the small amounts of aqueous obtained suffered partial oxidation during the weighing process, but the relative values would be unchanged. These experiments prove that

10. Cebione-Merck was used.

11. Müller, H. K., and Buschke, W.: Arch. f. Augenh. 108:592, 1934.

vitamin C is absorbed from the conjunctival sac into the aqueous humor. From the work done on alkaloids, it is known that this must take place through the cornea. If it can ever be shown that an artificial increase of the amount of vitamin C in the lens is of value in arresting the progress of a senile cataract, this experiment would indicate a means by which such increase can be effected, for by this method any vascular barrier to the passage of vitamin C from the blood into the anterior chamber is overcome. Such a condition is conceivable in cases of vascular sclerosis or of the inflammatory conditions leading to cataracta complicata. However, no satisfactory evidence has been reported that would indicate an effect of vitamin C on the progress of cataract.

The Effect of the Lens on Vitamin C.—It is well known that vitamin C in a neutral or alkaline solution is readily oxidized in air. Müller and Buschke ¹¹ demonstrated that aqueous humor containing a lens in vitro shows little diminution in its vitamin C content, while a similar amount

TABLE 2.—Concentration of Vitamin C in the Aqueous of a Treated and of a Control Eye

	Amount of Dye Reduced, Ce.	
	1st Reading	2d Reading
Rabbit 1*		
Aqueous from the eye treated with vitamin C.....	0.008	0.010
Aqueous from the control eye	0.005	0.004
	Vitamin C in Mg. per 100 Gm.	
Rabbit 2		
Aqueous from the eye treated with vitamin C.....	23	
Aqueous from the control eye	5	

* Prof. C. A. Farmer checked my results in this case.

of aqueous without a lens suffers a marked loss of this substance within a few hours. Green ¹² explained the preservation of vitamin C by the presence of an antioxidative substance in the tissues. The effect of the lens and of other substances on vitamin C is demonstrated in the following experiments.

EXPERIMENT 3.—Equal quantities of vitamin C were placed in two test tubes and to each 5 cc. of a phosphate buffer solution of p_H 7.4 was added. The lens of a guinea-pig was placed in one tube. The tubes were placed in an incubator at 37 C. The amounts of vitamin C recovered after two and a half and seventeen hours are given in table 3.

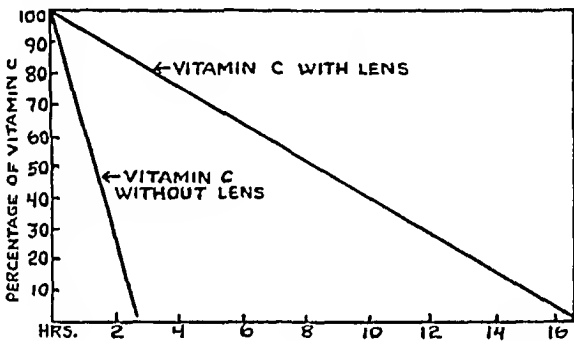
Again, equal quantities of vitamin C were placed in test tubes. To each was added 5 cc. of a phosphate buffer solution of p_H 7.4. A crystalline lens of a dog was added to one test tube, a small bead of glutathione to another, and the ash of a crystalline lens of a pig to another, and the final tube was used as a control

The whole series was placed in an incubator at 37 C. for one hour. The effect of these various substances on the amount of vitamin C is shown in table 4.

One sees from the foregoing experiments that vitamin C is preserved by the presence of crystalline lens or glutathione. The vitamin C does not escape from the lens into the solution to cause this difference. This is shown by a comparison of the amounts of this substance in the lens used in the solution and that of its fellow lens, the difference in this

TABLE 3.—Results Showing the Preservative Effect of the Lens on Vitamin C

	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered, Mg.	
		After 2½ Hr.	After 17 Hr.
Solution of guinea-pig lens.....	0.098	0.087 (89%)	0.011 (11%)
Control.....	0.098	0.003 (3%)	0



Graphic presentation of the preserving action of the guinea-pig lens on vitamin C.

TABLE 4.—Effect of Various Substances on Vitamin C

Substance Added	Amount of Vitamin C Added, Mg.	Amount of Vitamin C at End of 1 Hr., Mg.
Lens.....	0.098	0.018 (18%)
Control.....	0.098	0.005 (5%)
Glutathione.....	0.098	0.031 (32%)
Ash of lens.....	0.098	0.008 (8%)
Amount of cevitamic acid found in the first lens.....		0.028 mg.
Amount of cevitamic acid found in the fellow lens.....		0.032 mg.

case being only 0.004 mg. Because of the marked preserving action of glutathione, one might be led to believe that the preserving action of the lens depends on the presence of this substance. In order to determine whether or not the glutathione in the lens is the substance which preserves the vitamin C, the following experiment was undertaken.

EXPERIMENT 4.—A cataractous lens was extracted with a 2.5 per cent solution of meta-phosphoric acid. This extract gave no appreciable yield in reducing sub-

stances (glutathione or vitamin C) when titrated against thousandth-normal potassium iodate in a fine capillary microburet. Likewise, an immature cataractous lens was extracted which yielded 0.188 mg. of glutathione and 0.013 mg. of vitamin C. Of three test tubes, each containing 0.136 mg. of vitamin C, an aliquot portion of the extract of the first lens was added to one and an equal amount of the second lens extract to the second and the third was the control. A phosphate buffer solution of p_H 7.4 was added, and the whole was incubated at 37 C. The result showed that the preserving action of the mature cataract without glutathione was equal to that of the immature lens with a relatively large quantity of glutathione. Furthermore, the extract of a lens is quite as effective in preserving vitamin C as small pieces of tissues from other organs but is less effective than whole lens material.

TABLE 5.—*Preserving Action of Various Lenses on Vitamin C*

Animal	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered	
		After 1 Hr., Mg.	After 2 Hr., %
Dog.....	0.098	0.018	18
Dog.....	0.081	0.073	90
Rat.....	0.093	0.077	79
Guinea-pig.....	0.098	0.099	98
Rabbit.....	0.081	0.089	103 [*]

* Experimental error up to 5 per cent may explain this, yet the possibility exists that some vitamin C may be formed in the lens.

TABLE 6.—*A Comparison of the Preserving Action of Various Organs on Vitamin C*

	Amount of Vitamin C Added, Mg.	Amount of Vitamin C Recovered			
		After 1 Hr.		After 2 Hr.	
		Mg.	%	Mg.	%
Guinea-pig					
Lens.....	0.040	0.039	98	0.033	83
Liver.....	0.040	0.004	10	0.003	8
Spleen.....	0.040	0.004	10	0.003	8
Adrenal.....	0.040	0.005	20	0.006	15
Heart muscle.....	0.040	0.004	10	0.004	10
Control.....	0.040	0.004	10	0.002	5
Rat					
Lens.....	0.082	0.081	99	0.070	85
Liver.....	0.082	0.075	91	0.062	76
Spleen.....	0.082	0.039	48	0.031	37
Adrenal.....	0.082	0.013	16	0.012	16
Heart muscle.....	0.082	0.013	16	0.010	12
Control.....	0.082	0.005	10	0	0

From these experiments it is seen that although glutathione itself has a marked preserving action on vitamin C, a cataractous lens with no demonstrable glutathione is as effective as one with glutathione. This confirms Mawson's¹³ report that dialyzed tissues containing no glutathione were found to be effective in preserving vitamin C in alkaline solutions. The preserving action of various lenses on vitamin C is shown in table 5.

13. Mawson, C. A.: *Biochem. J.* 29:569, 1935.

In spite of the great differences in the size of the lens in the animals used in the experiments recorded in table 5, the preserving action is very similar and within certain limits seems to be independent of the amount of lens.

EXPERIMENT 5.—A comparison was made of the preserving action on vitamin C of equal weights of various organs of an animal in a phosphate buffer solution of p_H 7.4 at 37 C. (table 6).

One sees from this table that the lens exceeds by far the other organs in preserving vitamin C in the reduced form.

CONCLUSION

The remarkable preservative action of the lens on vitamin C as compared to other tissues seems to indicate its importance for maintaining the proper respiratory mechanism of the lens on which it is dependent for life. An interesting related fact is that the lens is also richer in glutathione and vitamin C than the other organs of the body. It would seem from the experimental work reported here that the antioxidative action of the lens on vitamin C does not depend on its content of glutathione and that still another factor must be present in the lens tissue which is important in preserving the respiratory mechanism of the lens. Although this work showed only the preservative action of vitamin C in the aqueous, it must be considered that such an effect takes place in the lens itself. This is borne out by the small loss of vitamin C sustained by the lens in the alkaline solution for one hour (experiment 4).

This action of the lens offers an explanation of the absence of the reduced form in the aqueous of the aphakic eye and the presence of only small amounts of the reversibly oxidized form of the vitamin C. With the removal of the lens, the mechanism for preventing the oxidation of the vitamin C in the aqueous which is a slightly alkaline solution (p_H 7.35) is lost. This explanation seems more plausible than that of Müller and Buschke¹⁴ and of Fischer,¹⁵ who explained this finding on the basis that the lens itself forms the vitamin C.

14. Müller, H. K., and Buschke, W.: *Arch. f. Augenh.* **108**:368, 1934.

15. Fischer, F. P.: *Klin. Wchnschr.* **13**:596, 1934.

CIRCULATION OF THE AQUEOUS

V. MECHANISM OF SCHLEMM'S CANAL

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In previous papers Dr. Pierce and I¹ have reported the experimental results in a systematic study regarding the mode of reabsorption of the various components of the intra-ocular fluid. These results may be summarized as follows: The reabsorption of water, of crystalloids and of colloids from the anterior chamber is in each instance effected by a separate and essentially unrelated mechanism. Colloids are removed by active phagocytosis on the part of the anterior layer of the cells of the iris, the endothelium. The removal of proteins is facilitated by the presence of proteolytic enzymes in the aqueous and the surrounding tissues. Crystalloid exchange takes place between the anterior chamber and the blood vessels of the iris by diffusion. The velocity of approach toward equilibrium in crystalloid content between the blood and the aqueous is controlled by the area of the iris (i. e., the state of contraction or dilatation of the pupil) and by the degree of hyperemia of the iris. In smaller part, some of the crystalloids of the aqueous are carried out of the anterior chamber along with the reabsorbed water.

In regard to the reabsorption of water we have shown, first, that this reabsorption takes place practically exclusively from the anterior chamber. Only the vessels of the iris and Schlemm's canal need, therefore, be considered as possibly concerned in this mechanism. Secondly, we showed that congestion of the anterior ocular vessels, whether active or passive, tended to facilitate, within limits, the reabsorption of water. It is evident that with a rise in the pressure within the vessels of the iris as a result of such congestion fluid should tend to enter the anterior chamber rather than to be withdrawn from it. From this we concluded that the iris plays only a subsidiary rôle in the intra-ocular fluid exchange. We were thus led, by a process of exclusion, to the conclusion that the chief mechanism concerned in the reabsorption of water from the eye is Schlemm's canal, and have suggested a possible mode of operation of the canal which would explain the seemingly para-

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1. Friedenwald, J. S., and Pierce, H. F.: Circulation of the Aqueous: II. Mechanism of Reabsorption of Fluid, *Arch. Ophth.* 8:9 (July) 1932; III. Reabsorption of Crystalloids, *ibid.* 10:449 (Oct.) 1933; IV. Reabsorption of Colloids, *ibid.* 14:599 (Oct.) 1935.

doxical fact that congestion of the anterior ocular vessels causes an increase in the rate of reabsorption of water. In the present paper it is proposed to formulate this hypothesis in greater detail and to present evidence which substantiates it.

The experiments of Duke-Elder² and those that we have reported indicate that the hydrostatic pressure within Schlemm's canal is equal to or slightly above that within the anterior chamber. It is impossible, therefore, to assume as has been done in the past that fluid enters Schlemm's canal by simple hydrostatic filtration. On the contrary, fluid can be drawn into the canal only by one or the other of two possible mechanisms: Either there is an active secretion of fluid into the lumen of the canal by its endothelial wall, or plasma proteins must be present within the canal in sufficient concentration to attract water osmotically from the anterior chamber into the canal. There is no evidence to indicate that a single layer of endothelium is capable of active secretion, and certainly such a vitalistic theory could not reasonably be suggested until the simpler physical possibilities have been excluded.

Since the hydrostatic pressure within the canal cannot far exceed that in the anterior chamber the concentration of plasma proteins required for the effective operation of the canal needs be quite small. Duke-Elder, for instance, estimates the pressure within the canal to be only 2 or 3 mm. of mercury higher than that in the anterior chamber. On the other hand, the osmotic pressure of the plasma proteins at their normal concentration is from 25 to 30 mm. of mercury. It follows that at equilibrium between the canal and the anterior chamber the plasma would be diluted about tenfold, and any increase in concentration of the plasma over this would entail the osmotic attraction of water from the anterior chamber into the canal.

It is evident that the hypothesis which we have formulated requires that plasma be supplied continuously to the canal from the blood vessels; that is to say, there is a constant stream of plasma into the canal from afferent arterioles just as there is a constant stream of plasma plus a greatly increased volume of water leaving the canal by efferent venules. Our conception, therefore, is that the canal lies between artery and vein as a specialized form of capillary sustaining a slight but steady through and through circulation of blood or plasma, rather than a blind outpouching of the episcleral veins as has hitherto been assumed.

The existence of afferent arteriolar connections with Schlemm's canal is a matter that may readily be demonstrated by histologic study, and it can be admitted at once that such afferent vessels are easily found. Indeed, it is somewhat surprising that they have not been discovered

2. Duke-Elder, W. S.: *Brit. J. Ophth.* **10**:513, 1926.

hitherto. Thus Maggiore³ in his classic studies on the anatomy of the canal describes only efferent connections, but his studies were based on specimens in which he had filled the canal with an injection mass introduced via the carotid arteries. It must be obvious that if the canal were merely a blind outpouching of the veins, as Maggiore concluded, one could not readily fill it by intravascular injection, for where could the normal fluid content run out? I have repeated Maggiore's experiments on rabbits and have found little difficulty in filling the canal with an injection mass introduced through the carotid arteries.

Sondermann⁴ in his studies on the anatomy of Schlemm's canal was chiefly concerned with problems of embryology. He showed that the anlage of the canal is a part of the iridociliary and intrascleral vascular net. Convinced that Schlemm's canal is a venous sinus, he concluded that the vessels afferent to and efferent from this vascular net are veins but furnished no proof of this statement.

Theobald⁵ in her beautiful reconstruction of Schlemm's canal confirmed the general anatomic findings of Maggiore and Sondermann and concluded: "There are no afferent vessels to the canal." But in the legend for her illustration, model 2-B, she said, "Note: The arteriole becoming a venule, and later receiving the upper collector from the canal of Schlemm." It should be clear, however, that the mere fact that a branch or continuation of an artery happens to be devoid of a muscular coat does not justify the assumption that the flow within that vessel normally goes toward the artery. It follows that the facts reported by Theobald do not warrant the categorical conclusion that vessels afferent to the canal are wholly lacking.

The data to be presented here are based on the study of serial sections of the anterior segments of four eyes, all essentially normal in regard to the portions under consideration. One of the four eyes was embedded in pyroxylin (celloidin) and cut at 15 microns. The remaining three were embedded in paraffin and cut at 7 microns. Since, with the important exceptions to be specially noted, the results of this study agree with those of Theobald and Maggiore, they will be presented in summary only. In all four eyes the canal was found to have three types of connections: (1) efferent venules, (2) afferent arterioles and (3) so-called inner canals.

THE EFFERENT VENULES

The observations in regard to the efferent venules are entirely in accord with those reported by Theobald. These vessels, upwards of twenty in number, join the canal with the intrascleral venous plexus

3. Maggiore: *Ann. di ottal. e clin. ocul.* **40**:317, 1917.

4. Sondermann: *Arch. f. Ophth.* **124**:521, 1930; **126**:341, 1931.

5. Theobald, Georgiana Dvorak: *Tr. Am. Ophth. Soc.* **32**:593, 1934.

which lies about midway in the thickness of the corneoscleral tissues at the limbus. They are lined by endothelium which lies directly on the fibers of the corneal stroma without intervening adventitia. Most often they are greatly flattened so that a cross-section may suggest the appearance of an oblique longitudinal section, and their dimensions can be accurately seen only when serial sections are studied. They reach their destination in the intrascleral venous plexus by a course which is sometimes straight but often rather tortuous. Occasionally they send a continuing branch to the episcleral vessels at the limbus.

THE AFFERENT ARTERIOLES

The afferent arterioles are much less numerous than the efferent venules and have hitherto apparently completely escaped notice. They occur as branches of the arterioles supplying the intrascleral plexus, but not every one of these arterioles possesses such a branch. These arterioles have been well described by both Maggiore and Theobald and are particularly well illustrated in the latter's paper. They arise from the ciliary arteries, enter the inner surface of the sclera at from 0.5 to 1 mm. behind the scleral spur and proceed obliquely forward and outward toward the intrascleral plexus at the limbus. Almost always each is accompanied by a small nerve and this, as well as the obvious thickness of the wall of the arteriole, serves to identify it. The number of these arterioles is about equal to that of the efferent venules, and most of them are not connected with Schlemm's canal. In each eye that I have examined there were, however, approximately from six to eight of these vessels that were connected with the canal, each by a short direct branch. The connection most often takes the form shown in figures 1 and 2. The arteriole is seen passing anterior to the canal and separated from it by about one third of the thickness of the cornea. Without deviating appreciably from its course it gives off a branch which, characteristically, forms a slightly acute angle in a retrograde direction with the parent stem and runs directly into the canal. Usually this connecting branch is devoid of any muscular coat and may therefore be readily confused with the efferent venule, but in some instances the wall of the vessel is appreciably thicker than that of the intrascleral venule and a minute twig of the nerve which accompanies the parent arteriole may sometimes be seen to branch off along with the connecting vessel.

These afferent arteriolar branches are usually circular in cross-section and have an internal diameter of only from 5 to 10 microns. The arteriole from which they branch is about one and a half to two times as large while the efferent venules, at least in their larger diameter, usually measure over 20 microns. The small diameter of these afferent

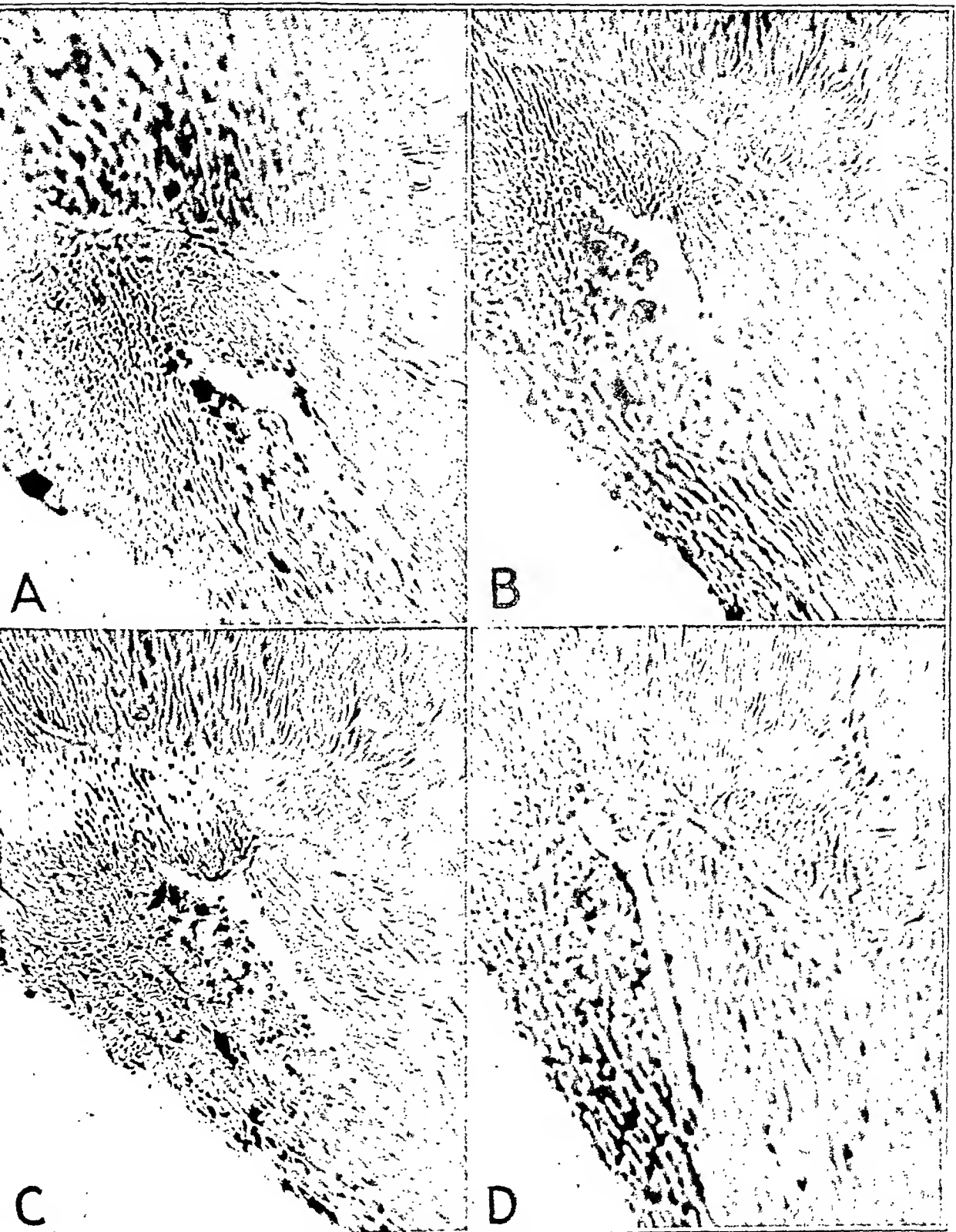


Fig. 1.—Successive serial sections through a vessel afferent to Schlemm's canal.

vessels, together with the slightly retrograde direction of their branching, helps to explain why red blood cells find their way into the canal in such small numbers. Neither of these facts, however, should interfere with the slow trickle of plasma from the arteriole into the canal. In fact, one may readily calculate that if the pressure in the arteriole is only from 5 to 10 mm. of mercury above that in the canal the amount of plasma flow required to keep up the normal rate of circulation of the intra-ocular fluid would be abundantly supplied. In their size, their number and their mode of connection with the canal these afferent vessels satisfy every requirement for the effective operation of Schlemm's canal according to the hypothesis outlined.

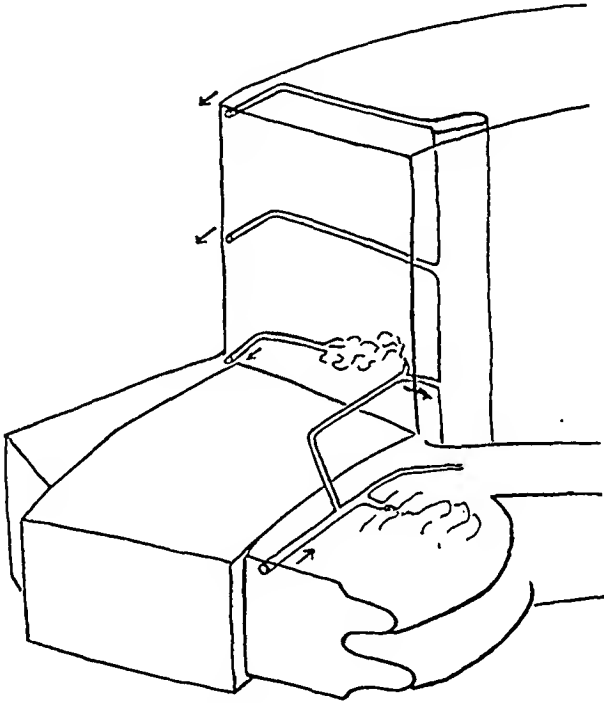


Fig. 2.—Schematic representation of the afferent and efferent connections of Schlemm's canal.

THE SO-CALLED INNER CANALS

Sondermann was the first to describe the outpouchings of Schlemm's canal between the trabeculae of the pectinate ligament. Both Sondermann and Theobald believe, on the basis of their histologic observations, that these outpouchings represent open connections between the canal and the anterior chamber, and they have, therefore, named these structures "inner canals." They have thus reopened the ancient controversy between Leber and Schwalbe on this point. It is to be remembered that the classic experiments of these two investigators and their many pupils, carried out with the most meticulous care, finally led to the conclusion

that there are no open connections between the canal and the anterior chamber. The results of these experiments are not lightly to be set aside. The walls of the so-called inner canals when visible consist of a single layer of very much flattened endothelium, so delicate as to appear threadlike even under high power microscopy. That these delicate walls could easily be broken and torn in the process of sectioning can hardly be doubted, and the attempt to follow in serial section the minute spaces which these walls enclose between the fibers of the pectinate ligament is open to grave error. The patency of these openings in life must, therefore, be seriously doubted, and Sondermann's own embryologic studies, which show that the canal embryologically is a plexus of completely walled vascular tubes, furnishes further doubt as to the existence of holes in these walls in the adult. If further support for the conclusions of Leber is required it can be found in the work of Maggiore. The latter, as noted earlier in this paper, found no difficulty in filling the canal with an opaque material injected intra-arterially, but in no case did he find that this injected material ran out of the canal into the anterior chamber. My own experience with Maggiore's technique is similar. The injected mass fills the canal fully but does not leak into the anterior chamber even if the pressure within the eye is reduced by corneal puncture. Finally, the existence of afferent arterioles communicating with the canal precludes the possibility of open connections between the canal and the anterior chamber, for if there were open connections plasma would necessarily leak from the canal into the anterior chamber and raise the protein concentration there. It must be concluded, therefore, that the so-called inner canals are actually blind pockets extending from the canal into the adjacent spaces of the pectinate ligament but separated from the anterior chamber in life by a continuous endothelial wall.

THE PRESENCE OF BLOOD IN SCHLEMM'S CANAL.

The conclusions which we have reached cast some new light on the long debated problem of whether or not the canal of Schlemm normally contains blood. Fuchs⁶ and Salzmann⁷ reported that they had observed the canal to be filled with blood in the living eye. Troncoso,⁸ on the other hand, found the canal when observed with the gonioscope to be pale and almost colorless. It is generally admitted that when the eye is congested the canal does in fact contain blood, which may even be demonstrable in histologic sections, though in sections of normal eyes red blood corpuscles are rarely seen within the canal. It should be

6. Fuchs, E.: *Ber. ü. d. Versamml. d. ophth. Gesellsch.* **28**:136, 1900.

7. Salzmann: *Ztschr. f. Augenh.* **31**:1, 1914; **34**:26, 1915.

8. Troncoso, M. U.: *Am. J. Ophth.* **8**:433, 1925.

remembered, however, that when an eye is removed for histologic study the pressure in the vessels connected with Schlemm's canal necessarily falls promptly to zero, and that any additional fluid which may filter into the canal under the influence of the still appreciable intra-ocular pressure must tend to flush out the canal and clear it of the few red blood corpuscles which it might have contained during life. Even if the eye is placed as rapidly as possible in a fixing solution, such filtration should continue for some time because time is required for the fixative to penetrate through the sclera to the canal. A much less efficient mechanism of a similar kind is at hand to empty the retinal arteries of their blood, and one does, in fact, often find them practically empty in histologic sections, but one does not, on that account, conclude that they are empty of blood during life.

From the point of view of the present investigation it is clear that the blood normally entering the canal would be greatly diluted with aqueous, and that the corpuscular content may be expected to be very small if a skimming of the plasma should take place at the origin of the afferent arteriolar branch. It is not surprising, therefore, that the region of the canal when viewed with the gonioscope is practically devoid of color.

OBSERVATIONS WITH POSSIBLE BEARING ON THE THEORY OF GLAUCOMA

In conclusion one may consider what possible bearings the present conception of the mode of operation of Schlemm's canal may have on the theory of glaucoma. It has been shown that hyperemia of the vascular bed with which the canal is connected increases the rate of reabsorption of the intra-ocular fluid. One may conclude that a decrease in the flow of plasma through the afferent arterioles into the canal would decrease the efficiency of the canal in removing fluid from the eye and lead to a rise in the intra-ocular tension. It would be of interest, therefore, to know whether sclerotic changes are to be found frequently in the afferent arterioles of eyes affected with primary simple glaucoma. Particularly it would be important to know whether such changes are found early in eyes with primary simple glaucoma.

The first question can be readily answered in the affirmative. In eyes showing advanced chronic glaucoma, which form the great bulk of those available for histologic study, the afferent arterioles and the arterioles of the intrascleral plexus of which they are branches commonly show marked hyaline degeneration of their walls with reduction in their lumens and even occasional thrombotic occlusion. A few of the changes found are illustrated in figures 3 and 4. But one cannot immediately assume that these arteriolar changes are primary to the glaucoma.

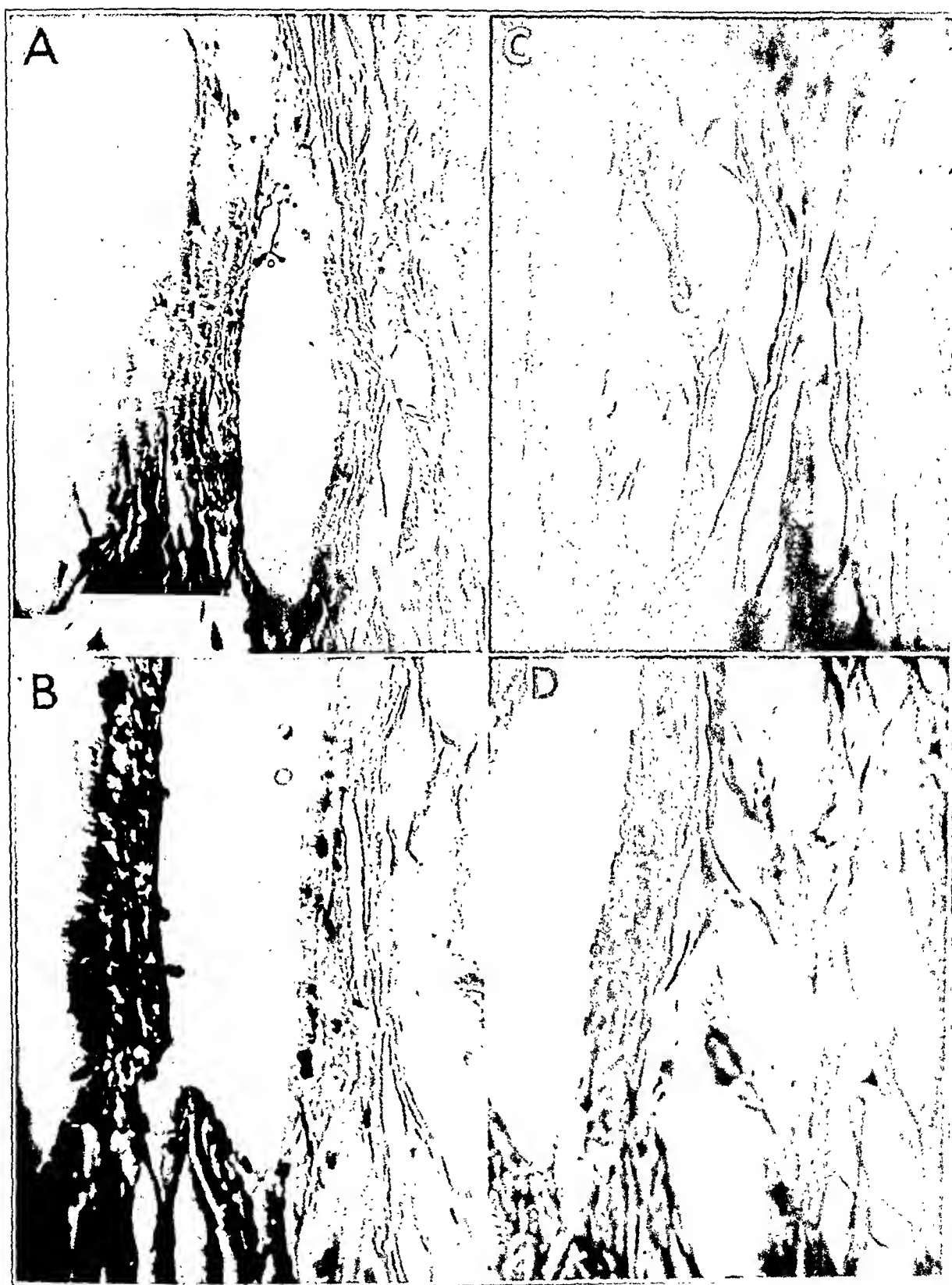


Fig. 3.—Sections from a case of early glaucoma simplex: *A*, Schlemm's canal with normal pectinate ligament; *B*, Schlemm's canal with normal "inner canal"; *C*, and *D*, hyaline degeneration of arteriodes afferent to Schlemm's canal.

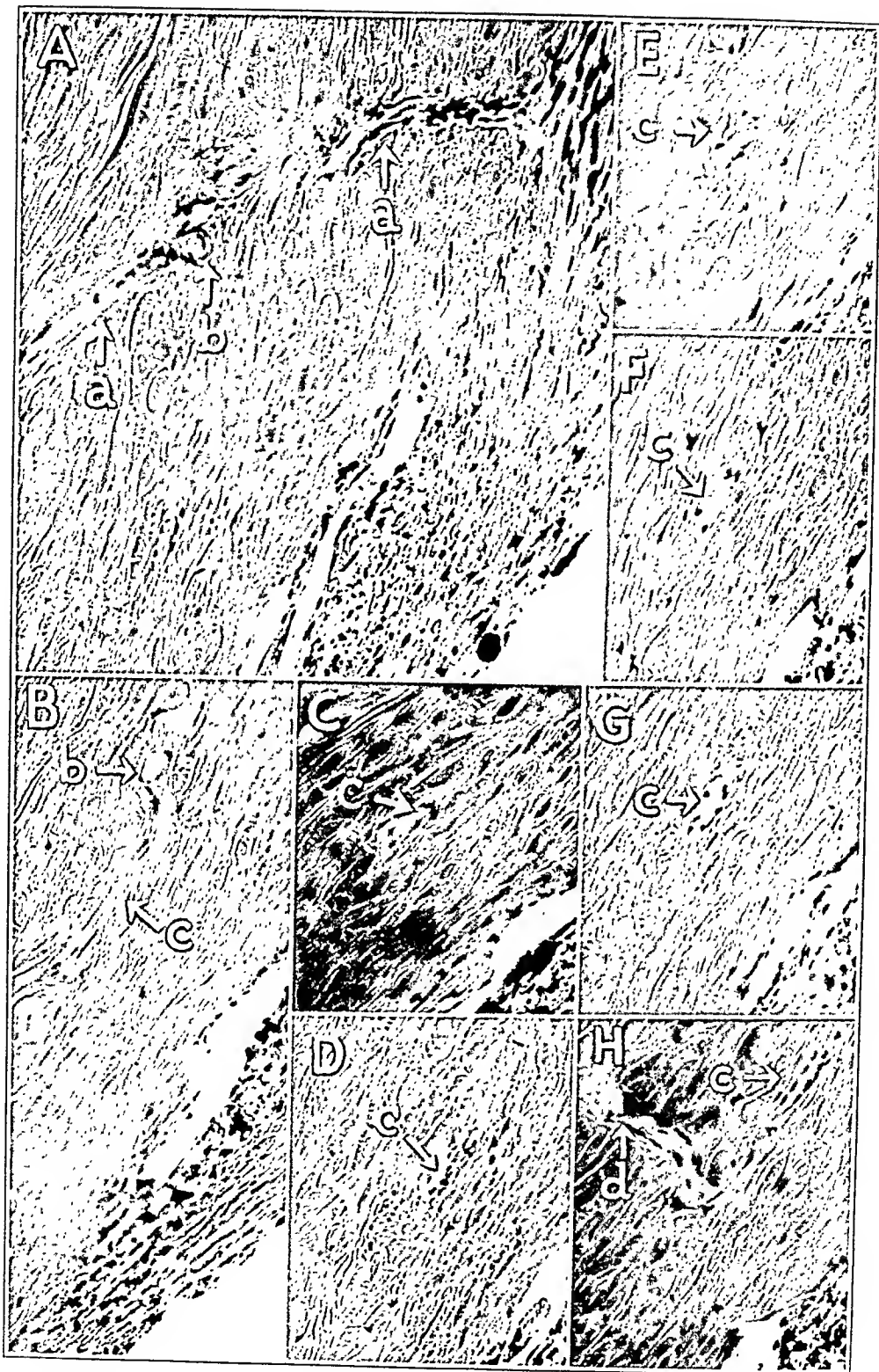


Fig. 4.—Photomicrographs selected from seventy-five successive serial sections in a case of absolute glaucoma in which degenerative changes had occurred in the vessels connected with Schlemm's canal. Compare with figure 6. An artery, *a-a*, which arises with its accompanying nerve from the ciliary body is seen in *A*. This gives off a smaller branch, *b*, which runs perpendicularly to the plane of the sections. In *B* this branch gives off a branch, *c*, which can be followed through *C* to *H*. In *E* and *F* the wall of *c* is necrotic and the lumen filled with a thrombus. In *H*, *c* bifurcates into *d* and *e* (*e* shown in fig. 5 *A*).

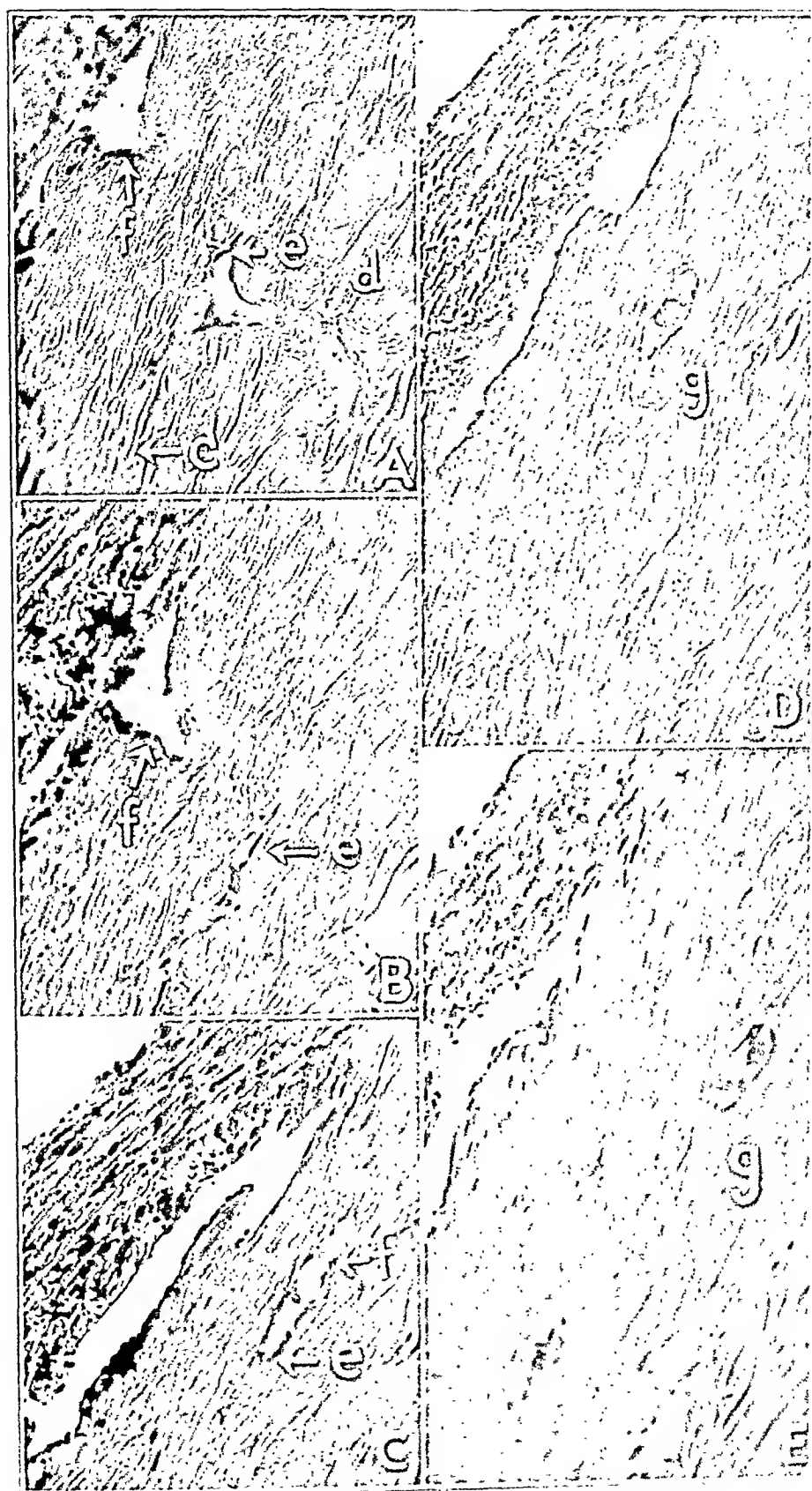


Fig. 5.—Branch *c* connects with an outgrowth of Schlemm's canal *d* in *A*, a branch arising at this junction, *e*, shows a proliferation of its outgrowth in *B* and *E*.

Up to the present only one case of early and uncomplicated simple chronic glaucoma has been available to me for histologic study. The patient was a white man of 73 who had been known to have glaucoma for eighteen months prior to his death. The highest recorded intra-ocular tension had been 26 mm. of mercury (Schiotz) in each eye, and under miotics the tension was reduced to 12 mm. The anterior chambers were of normal depth, the visual acuity was normal in each eye, but the optic disks showed marked glaucomatous excavations. The peripheral regions of the fields were normal, but one eye showed an enlargement of the blindspot of the Seidel type, while the other showed a marked Bjerrum scotoma. Three weeks after my last examination of this patient he died of pyelonephritis following prostatectomy.

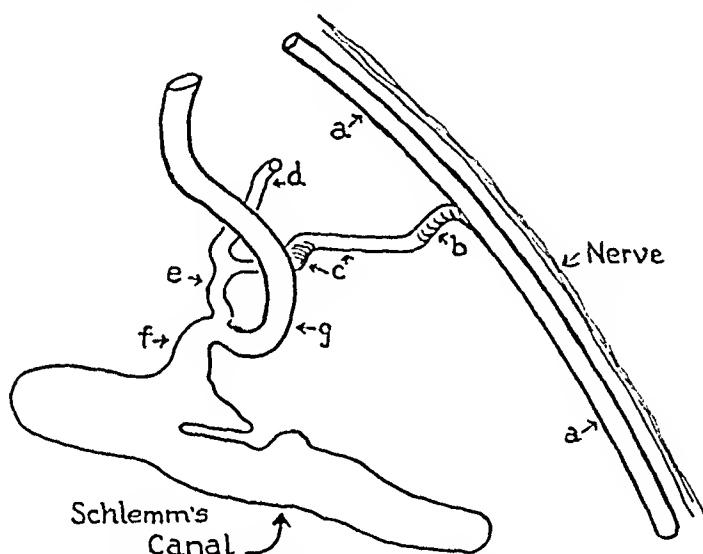


Fig. 6.—Schematic drawing showing the connections of the vessels shown in figures 4 and 5.

Examination of his eyes post mortem revealed the glaucomatous excavation of the disks with slight optic atrophy. There were no peripheral anterior synechiae. The ciliary body was normal. Schlemm's canal was patent, and the pectinate ligament was not sclerosed (fig. 5). The afferent arterioles of the canal showed marked hyaline thickening of their walls and reduction of their lumens. The degree of sclerosis of these vessels considerably exceeded that of the other intra-ocular arterioles. Aside from a moderate arcus senilis and some cystic degeneration of the anterior part of the retina, no other changes were found in the eyes.

It should be made abundantly clear that the observations in this single case, even though supported by similar observations in eyes showing the more advanced stages of the disease, are wholly inadequate to confirm the hypothesis as to the pathogenesis of chronic glaucoma which I have outlined. Furthermore, it cannot be assumed that there is

only one possible cause for the disease picture which is classified as chronic simple glaucoma. Nevertheless, it seems worth while to formulate this hypothesis and to report the confirmatory evidence, feeble though it is, because cases of uncomplicated early glaucoma so rarely present themselves for histologic study and the opportunity for confirmation or refutation of this hypothesis may fall into other hands than mine.

SUMMARY AND CONCLUSIONS

Schlemm's canal is not connected with the anterior chamber by any open channels or pores. It is connected with the intrascleral vascular plexus at the limbus by both afferent arterioles and efferent venules, the latter about four times as numerous as the former.

The effects of a continuous flow of plasma from the afferent arteriole into the canal and of the dilution of the plasma within the canal with water osmotically attracted from the anterior chamber are discussed. It is shown that, within limits, an increase in the flow of plasma into the canal leads to an increase in the rate of absorption of the aqueous and a lowering of intra-ocular tension. Similarly a reduction in the flow of plasma into the canal would be expected to lead to a decrease in the rate of absorption of the aqueous and a rise in the intra-ocular tension. It is suggested that in some cases of chronic simple glaucoma the primary lesion may be a sclerotic occlusion of the afferent arterioles. Such sclerotic changes were found regularly in cases of advanced glaucoma and also in one case of early uncomplicated chronic simple glaucoma.

ISAAC HAYS

PIONEER AMERICAN OPHTHALMOLOGIST

NATHAN FLAXMAN, M.D.

CHICAGO

Isaac Hays, the son of Samuel and Richea Gratz Hays, was born in Philadelphia on July 5, 1796. His father, a wealthy merchant engaged in the East India trade, brought up his family with all the culture and luxury which his means enabled him to command. Isaac acquired from the refinements of his early home life the courteous and affable manner which later distinguished him.

The early education of the youthful Hays was obtained as a pupil of the Rev. Dr. Samuel B. Wylie, an eminent divine and classical scholar of Philadelphia. At the age of 16 Isaac entered the University of Pennsylvania. Here he obtained the degree of Bachelor of Arts in 1816. The elder Hays desired to have his oldest son succeed him in the mercantile business. Although young Isaac yearned for a professional career, he obeyed his father and worked one year in the counting house. He then abandoned his business career since he had no taste for such a life. His father not only agreed to this but helped to start him in the medical profession.

The motives that inclined the 21 year old Isaac toward the study of medicine revolved about one man—Dr. Nathaniel Chapman. In 1817 Isaac became an office pupil of this outstanding practitioner. The man himself attracted the eager student. Chapman was the medical man of his day, a Virginian by birth, wealthy, cultured, with a magnetic personality and of the highest social standing. Everybody knew Chapman, and he knew everybody. The friendship formed between preceptor and pupil during those early days endured and grew firmer over a period of thirty-six years.

During the period that he was an office pupil of Chapman, Hays attended the medical school of the University of Pennsylvania. He obtained his doctor's degree from this institution in 1820. The faculty at that time consisted of Physick, Wistar, James, Dorsey, Coxe, Chapman, Hare and Gibson. Hays entered practice while he lived at the northeast corner of Eighth and Sansom streets. Exactly from whom or how he became interested primarily in the diseases of the eye was not apparent. At the time he entered the practice of medicine in Philadelphia in 1820, George Frick of Baltimore was the only doctor in the

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country limiting his practice to ophthalmology. The few general surgeons who were attempting to do ocular work were poorly equipped. Their general knowledge of anatomy of the eye was good, but their conception of pathology was poor. The methods of examination were limited to inspection with the unaided eye and to palpation. Hays was an ambitious young man, inspired by the success of Chapman and desirous of making a name for himself in an untouched and unlimited field. His deliberate nature, mathematical interest and precise work well fitted him for the practice of ophthalmology.

The first textbook on the diseases of the eye printed in the United States was an American edition of Saunders' text which was published in Philadelphia in 1821. Hays consumed the material in this book from cover to cover. Most of his time was devoted to laying a firm foundation for future work, and much leisure was spent in the study of ocular conditions. He was appointed to the staff of the first ophthalmic dispensary founded in Philadelphia, the third in the United States in the same year. This dispensary was in existence until 1825. When the fourth ophthalmic hospital, the Pennsylvania Infirmary for Diseases of the Eye and Ear, was opened on Feb. 8, 1822, he was appointed one of the surgeons; the others were Robert Griffith, George Wood and John Bell. In that institution he obtained the actual clinical experience necessary to balance the vast amount of theoretical knowledge gained in his first two years in practice. The first work of an American on diseases of the eye, George Frick, on diseases of the eye, was published in 1824. An extended review of this book, written by Hays, appeared in the 1825 issue of the *Philadelphia Journal of the Medical and Physical Sciences*, which was edited by Chapman.

The Academy of Natural Sciences elected Hays to membership in 1818, and shortly afterward he was made a member and chairman of the Publishing Committee, in which office he was earnest and indefatigable. His first medical paper, entitled "The Forces by which the Blood is Circulated," was delivered as a lecture to the Academy of Natural Sciences in 1823 and was published as the leading article in the *Philadelphia Journal of the Medical and Physical Sciences* in 1826. This paper summarized the successive steps in a complete knowledge of the subject up to that time.

During those early years in practice Hays gained and held the friendship of a number of practitioners of his own age. All of them, Samuel Jackson, Franklin Bache, Charles Meigs, Rene LaRoche, George McClellan, Hugh Hodge, John Mitchell, Francis Condie and George Woods, were hard-working, ambitious men, devoted students like Hays, good writers and fine teachers, with scholarly tastes, refined manners and cultivated intellects. These men, constantly acting and reacting on one another, stimulated each other's ambitions.

In the few years before other important decisions had to be made, Hays laid the foundation of high repute as an ophthalmic surgeon. He intended to publish a series of papers on various ocular diseases. This was very evident with the publication of the first article, entitled "Observations of the Inflammations of the Conjunctiva."¹ In the same year another long article appeared, "Inflammations of the Sclera,"² and this was followed by an extensive treatise on "The Pathology and Treatment of Iritis."³ The nature of these articles indicates that Hays began a systematic study of his own observations on the clinical material seen in the Pennsylvania Infirmary and the Philadelphia Dispensary.

Hays showed unusual ability in another line of work, medical journalism. While his extensive work on diseases of the eye was appearing in print, he had to make the most momentous decision that could possibly confront a rising young practitioner of 30 years. When he became associated with Chapman, Dewees and Godman in the editorial management of the *Philadelphia Journal of the Medical and Physical Sciences* in 1826, and, when late in 1827 he became the founder and editor of the *American Journal of the Medical Sciences*, Hays stood at the brink of two tremendously fertile and undeveloped fields. Well qualified in both, he firmly believed that by devoting the major portion of his time to medical journalism some improvement in ophthalmology would result. Therefore he resigned from the staff of the Pennsylvania Infirmary in 1827 and concentrated his efforts in placing the first national medical journal on a successful basis.

When Dewees' textbook on the "Practice of Medicine" appeared in 1830, the chapter on the diseases of the eye was contributed by Hays. This chapter covered a concise account of the anatomy, physiology and pathology of the eye and the treatment of its diseases in about 70 pages. Dewees considered Hays the best and most suitable person to write that chapter for the book, and in the preface he not only mentioned this but alluded to Hays' long and attentive study of the subject together with his experience.

In 1830 James Wills of Philadelphia left a bequest to found a hospital for the indigent blind and lame. A building was erected on Race Street between Eighteenth and Nineteenth streets. The hospital was opened on March 3, 1834, and continued its work on the same site to the present day. The first staff was composed of Isaac Parrish, Squier Littell, ~~George Fox and Isaac Hays~~. ~~After a few years the work of~~ the Wills Hospital became entirely limited to the diseases of the eye. Hays was on service three months and off service three months, alternating with George Fox. For twenty years, from 1834 to 1854, Hays

1. Philadelphia J. M. & Phys. Sc. **13**:84, 1826.

2. Philadelphia J. M. & Phys. Sc. **13**:211, 1826.

3. Philadelphia J. M. & Phys. Sc. **14**:217, 1827.

served this institution. During this period he contributed reports on the cases seen in his service in this hospital to the *American Journal of the Medical Sciences*. The other attending ophthalmic surgeons did likewise, and this journal published clinical material on the diseases of the eye continuously, which, in addition to the selected abstracts on ophthalmology that Hays contributed to that section of the magazine, was the only ophthalmologic literature uninterruptedly available to all practitioners for over forty years.

Two outstanding contributions to ophthalmology were made by Hays in the case reports from the Wills Hospital. He first observed and reported noncongenital color blindness as a pathologic condition—the case of Mary Bishop, which ended in recovery.⁴ Although he did not record the first case of astigmatism in America, and the fifth in the world, until 1854, he noted this condition as early as 1839. This same article of 1840 contains references to cases of astigmatism. In 1843 he edited an American edition of Lawrence's treatise on diseases of the eye. A letter from Lawrence acknowledging a copy of the volume contained the following paragraph:

I return you my best thanks for the book, and at the same time beg to express to you how highly I am gratified at finding that you not only think my work worthy of republication in America, but that you should have condescended to act as editor, and to enrich my treatise with those valuable additions which might well have constituted a separate publication. I feel that I could not have received a higher compliment, and I shall always hold the circumstance in grateful remembrance.

The preparations of the successive editions of this work was to Hays a delightful task not only because it associated his name with that of perhaps the outstanding ophthalmic surgeon in England but because it gave him an opportunity, as he himself said in the preface of the second edition, of presenting the results of his own experience derived from more than a quarter of a century's devotion to the subject, during which period he had been attached to a public institution for the treatment of the diseases of the eye. In the third edition, in 1854, he first recorded the correction of astigmatism in two cases, the cylindric glasses having been ground by McAllister,⁵ the Philadelphia optician, in 1853. Bünstead carried on this work in New York and Hasket Derby in Boston. On receiving a copy of the third edition of his book, Lawrence wrote a letter of thanks to Hays:

I feel it a particularly fortunate circumstance that one so thoroughly conversant with the whole subject, and so used to literary composition, should have undertaken the troublesome task of making these additions to my treatise, pub-

4. *Am. J. M. Sc.* 26:277 (Aug.) 1840.

5. Personal communication to the author from Dr. G. E. de Schweinitz.

lished in 1840, which are necessary to bring it up to the present state of knowledge and should have conferred upon it the new and interesting feature of so many beautifully executed pictorial illustrations.

An instrument, combining the advantages of a knife and a needle, for the discission of hard cataracts was devised by Hays. Although it became obsolete later, the instrument was at the time much employed by American oculists. This needle-knife was used for the operation of cataract "by solution or absorption," and Hubbell said that "not only did this instrument admirably serve the purpose for which it was designed by Dr. Hays, but it is still an excellent knife for discission of after-cataract." Hays also edited, with notes, the American editions of T. Wharton Jones' "Principles and Practice of Ophthalmic Surgery" in 1847.

These contributions certainly suggest that Hays kept his early interest in ophthalmic problems. When the tremendous task of editing the national quarterly, and later the associated monthly journal, allowed some leisure time, he delved into his first love. The failure of Hays to become the outstanding ophthalmologist in the United States was due to the fact that he never became a teacher of that branch of medicine. This was not on account of his natural timidity before an audience. It was purely an accidental occurrence. He was to deliver the introductory lectures on the diseases of the eye and ear at the Philadelphia Anatomical Rooms in College Avenue, later called Chant Street, in the winter of 1826, but the lectures were never given. The removal of Godman, the main projector of the enterprise and the lecturer on anatomy and operative surgery, to New York and Hays' acceptance of the editorial office with the *Philadelphia Journal of the Medical and Physical Sciences* probably prevented him from becoming the most thorough and accurate teacher of his day. In later years whatever he was called on to say was to the point and tersely expressed. He impressed his hearers with a sincere and balanced conviction of his opinions.

At the age of 38 years he married Sarah Minus of Savannah, Ga., and this happy union was eventually blessed with four children. The peace and content of his home contributed much to the diligent and steady performance of his increasing literary work. He was rarely out of Philadelphia, even in the summer, and he did not have a vacation for twenty years after he became editor of the *American Journal of the Medical Sciences*. Hays was proportionately built and had blue eyes and a well formed head with a finely chiseled profile and a beaming and benevolent countenance. In his manners he was emphatically a gentleman of the old school, bland, gentle and dignified, with a sweet and subdued voice and a warm sympathizing heart. He was an early riser and seldom required more than five or six hours of sleep. His jour-

nalistic work was usually done in the early morning or in the evening, thus leaving him the remainder of the day for his professional, literary and public duties.

The life and work of Hays—these were synonymous to him—aside from ophthalmology, consisted of editing the *American Journal of the Medical Sciences* for fifty-two years and affiliated journals for thirty-six years and of his tremendous effort in behalf of the medical profession, individually and collectively.

In 1854, at the age of 58 years, Hays gave up all clinical work to devote himself to literary work. This arrangement was only for a short time, as he soon reentered private practice. Black stated that in 1858 in ophthalmology Hays and Squier Littell did little or no other work in Philadelphia; all the general surgeons did ophthalmologic work. In the sunset of Hays' life the American Ophthalmological Society was founded, in 1864, and Herman Knapp began the *Archives of Ophthalmology*, in 1869. When the Philadelphia Ophthalmological Society was founded, in 1870, Hays was elected its first president, at the age of 74 years. His son, I. Minus, was associated with him in both the editorial and the ophthalmologic work from 1869 to 1879.

With the advance of age, Hays' strength gradually gave way. His mind was active and still carried keen interest in all his work. In February 1879, influenza was epidemic in Philadelphia, and he was one of the sufferers from this malady. He outlived this attack but never rallied from its effects or regained his strength, being confined to his residence at 1525 Locust Street. On April 12, 1879, Isaac Hays passed from this life, one of devotion and beauty, at the venerable age of 83.

No monuments were raised to this man who devoted his lifetime to the interests of his profession. In every medical library of the world his remarkable work stands—103 volumes of the first and finest national medical quarterly. This journal was more than just a magazine to him. It was a living thing, throbbing with the ideas and thoughts of ambitious, hard-working, struggling practitioners everywhere. His relation to the men who contributed to this journal and what the publication of papers in it meant to these persons was best expressed, indirectly, by J. Marion Sims. While suffering from severe diarrhea which had forced him to leave the South, Sims went to Philadelphia in July 1852 to see some friends, as he felt he was going to die.

When I saw that I could not recover, I sent for my friend Dr. Isaac Hays to come and see me. He came very promptly. I explained to him the condition of my affairs, and said to him that I felt I was going to die. He said that he thought I had better take cod-liver oil, and not to give up.

When Sims sought and obtained a home at 89 Madison Avenue in New York in September 1853, with little money on hand, no friends,

no influence, no health and nothing to commend him to business, he said :

Fortunately I had published my article on the treatment of Vesico-vaginal fistula a year before that, in the American Journal of the Medical Sciences, and the doctors had read it everywhere, and were very much surprised at the claims set up of rendering this troublesome and loathsome affection easily and successfully cured. They hardly believed it. Whenever I was introduced to any of the doctors, they all knew who I was by that article, and by my previous contributions to the medical literature.

All of Hays' work has endured. His effect on American ophthalmology, medical journalism and organization can be noted in the firm foundation on which the profession stands. His pioneer work in the ocular field has been considered. In his efforts in behalf of the medical profession Hays can be compared with the most prominent figure in American medicine, William Osler. Three men, Benjamin Rush, Isaac Hays and William Osler, stand foremost in this country in the three hundred years of its medical growth. The first two were native Philadelphians, and the third was an adopted son. Hays lacked the sparkling personality of Osler, but in his quiet unassuming way the work of fifty-two years, steady unrelenting labor, shaped American medicine to conform with his high ideals of the ethics and dignity of such noble work.

The selection of Hays by the editors and publishers of the *Philadelphia Journal of the Medical and Physical Sciences* in 1826 to the staff of the journal was neither an accident nor an experiment. Isaac Lea, a member of the house that published this journal, was well acquainted with Hays' work in editing the *Journal of the Academy of Natural Sciences*. The *Philadelphia Journal of the Medical and Physical Sciences* originated in 1820 with Chapman as editor, and in 1825 William P. Dewees and John Godman were added to the staff. In the following year Godman moved to New York. The other editors were unable to devote their time to the interests of the journal because of increasing professional activities. It became necessary to select another editor, and the friendship of Hays with Chapman and Dewees and his acquaintanceship with Godman, along with the knowledge of his work by the publisher, Lea, made Hays the unanimous choice for the position. An announcement to this effect, prepared by Godman, appeared in the *Philadelphia Journal of the Medical and Physical Sciences* :

The Publishers, with entire approbation of the Editors, announce with pleasure that they have secured the valuable assistance of Isaac Hays, M.D., who henceforth will co-operate in conducting the Journal. Independent of this gentleman's practical experience and zealous devotion to the interest and honor of the profession, we consider him as a great acquisition, on account of his long experience and well earned reputation in the management of such a publication. The Journal of the Academy of Natural Sciences owes a very large portion of its success to

his exertions and attention to the business of its publication. In adding him to the editors we give an ample pledge of our determination to make every effort to merit a continuance of the liberal support it has uniformly received.

The name of Isaac Hays appeared as one of the editors on the cover of the second number of the thirteenth volume of the *Philadelphia Journal of the Medical and Physical Sciences* in February 1826. By the end of the year he was the sole editor, as all his associates had retired. The Philadelphia journal had been created by Chapman in a spirit of indignation at the remark of Sidney Smith made in an essay in the *Edinburgh Review*: "In the four quarters of the globe, who reads an American Book? Or goes to an American play? Or looks at an American Statue? What does the world yet owe to American Physicians or Surgeons?" To emphasize his feelings, Chapman, as long as he was editor, quoted that arousing remark on the title page.

Hays did nothing by excess or impulse. His was a well balanced character. The position of American medical journalism in 1827, thirty years after its birth, was very uncertain. Hays was aware of this. The *Philadelphia Journal of the Medical and Physical Sciences* was the thirteenth medical magazine to be published in the United States since 1797. Ten of the thirteen journals, all local in character, had expired prematurely by the time that Hays became an editor. The ninth journal, founded in 1812, the *New England Journal of Medicine and Surgery*, and the twelfth, founded in 1818, the *Medical Recorder*, as the *Philadelphia Monthly Journal of Medicine and Surgery* was called, were the only ones in print at that time besides the *Philadelphia Journal of the Medical and Physical Sciences*. Hays also was of the opinion that the position of a profession was reflected in its magazines. Therefore, he laid aside the local character of the *Philadelphia Journal of the Medical and Physical Sciences*, combined it with the *Medical Recorder*, discarded Sidney Smith's remark from the title page, gave the new quarterly the name of the *American Journal of the Medical Sciences*, selected a group of thirty-nine collaborators representing many of the best and prominent men in the various sections in the country and issued the first number of the first national medical journal in November 1827.

His platform was to advance the interests of medical science, to foster and develop native talent, to disseminate medical knowledge, to elevate the character and dignity of the profession and to supply a want deeply felt by the American practitioner. Anonymous contributions, as well as all personalities, he scrupulously excluded. Each number of the *American Journal of the Medical Sciences* was issued under three separate headings: the department for original communications,

a review and biographical department and a perioscopic department devoted to a condensed account of the recent progress in medical science in the entire world.

As an editor Hays possessed an enlightened, unbiased and deliberate judgment. His manner invited confidence. He had a firmness that was proof equally against the allurements of designing flattery and the exactions of overweening self-esteem as well as against the dictation of ill advised counselors and the injustice of hostile criticism. These influences never disturbed the serenity of his feelings. He did what he felt obliged to do, never rashly or impulsively, and not with a zeal that was blind to the right of judgment in others. He kept before him the fact that errors may be propagated fanatically by men who are honest in their convictions and aims. It is almost without example that this leading medical periodical which he edited for more than half a century was never known as the organ of a party and never contained an editorial line that betrayed personal animosity. Until 1841 Hays edited the *American Journal of the Medical Sciences* with the aid of collaborators. In that year he became the sole editor, his name appearing for the first time on the title page of a new series of the quarterly. It became affectionately known by practitioners everywhere as the "Hays Journal." Many of the early bound issues have that title instead of the real name of the journal imprinted on the binding. For the next twenty-eight years he carried on all of the editorial work alone.

The *Cholera Gazette* of 1832, a weekly, was edited by Hays. Cholera appeared in Europe in that year and spread to Canada and New York. The Philadelphia Board of Health appealed to the local medical society, and a committee was appointed to investigate the matter. The reports of this committee were published in the *Cholera Gazette*. This bulletin was the attempt of medicine to organize as effectively as possible to stop the plague by reaching the physicians and the public with all possible information on the subject.

The dignity and gravity of the quarterly journal prevented it from containing discussions on the problems which concerned the medical profession not directly connected with the art and science of medicine. Therefore, in 1843 Hays founded the *Medical News* as a monthly journal to keep physicians informed of all that transpired of general interest in the various medical societies and colleges of the country, and especially of everything that tended to advance or retard the course of medical education. Part of its columns were also devoted to exposing quackery in all phases and forms. In pursuit of this reformatory work Hays only once found himself called on to answer for what he had edited. In 1846 a suit was brought against him by the author of a quack remedy for having declared in the *Medical News* that the pamphlet describing this medicine "displays quackery in its most unblushing

and undisguised form." The case was tried in the Supreme Court of the state, and the jury sustained the criticism of Hays by merely awarding nominal damages to his assailant. The *Bulletin of Medical Science*, edited by John Bell, commented on the case as follows: "We must not let the opportunity pass without recording the obligations under which the profession lies to Dr. Hays for his continual exposure of quackery and medical delusions." The *Medical News*, which afforded a ready field for the display of personal feelings, never exhibited them during the years that Hays conducted it, not even at times when there was sufficient ground in the misconduct of others to justify the severest type of criticism.

In 1874 he began the publication of the *Monthly Abstract of Medical Science*, the forerunner of subsequent journals of abstracts, but this was merged with the *Medical News* the year after the death of its founder. Gross made the prediction at the time of Hays' death that when the history of American medical literature is written an important place will be assigned to this, the greatest and most gifted medical journalist of the nineteenth century.

The *Medical News*, from its inception in 1843, continually devoted space to the striving of individuals for better medical education and to the work of medical societies to improve the teaching and licensing of practitioners. The part Hays took in the organization of the American Medical Association was directly in line with his editorial ideas on the subject. Early in 1846, when the Medical Society of New York sent out the call for the first convention, he was sent as a delegate by the Philadelphia Medical Society. He took an active part in the proceedings that opened the first meeting on May 5, 1846, in New York. As a member of the committee appointed to bring the subject of medical education before the convention, he presented the resolutions that proposed to create a national medical association and to adopt a uniform and elevated standard of requirements for the degree of Doctor of Medicine so that young men before being received as medical students would obtain suitable preliminary education and that the medical profession in the United States would be governed by the same code of medical ethics. The following account of the origin of these resolutions was noted in an original manuscript of Hays:

The idea of taking advantage of that occasion to form a National Medical Association is due, I have reason to believe to Dr. A. Stillé of Philadelphia. He consulted one of his colleagues (Dr. Hays) in regard to a series of resolutions which he had prepared to offer for the consideration of the convention; but as no opportunity for his offering them had presented itself, so great was the disorder on the first day of the meeting, a colleague (Dr. Hays), who had been appointed one of the committee to prepare the business for the next day, asked to be allowed to bring them before that committee and try to obtain the recommendation of the

passage of these resolutions. With some difficulty this was obtained, and the resolutions adopted by the convention were essentially the same as those prepared by Prof. Stillé.

These subjects were referred to several committees, and Hays was appointed a member of the one to prepare a code of medical ethics. In the meanwhile the medical journals published the proceedings of the convention in New York. This helped bring the subject to the attention of a large number of the members of the profession. Hays, as chairman of the Committee of Arrangements, welcomed the delegates to the meeting in Philadelphia on May 5, 1847, at the organization of the association. His report on the subject of medical ethics which was the work of the entire committee was very full and explicit. He stated that justice required that some explanatory remarks should accompany the code which was unanimously adopted by the convention:

The members of the Convention would not fail to recognize in parts of it, expressions with which they were familiar. On examining a great number of codes of ethics adopted by different societies in the United States, it was found that they were all based on that by Dr. Percival, and that the phrases of this writer were preserved, to a considerable extent in all of them. Believing that language so often examined and adopted must possess the greatest of merits for such a document as the present, clearness and precision, and having no ambition for the honors of authorship, the Committee which prepared this code have followed a similar course, and have carefully preserved the words of Percival whenever they convey the precepts it is wished to inculcate. A few of the sections are in the words of the late Dr. Rush, and one or two sentences are from other writers. But in all cases, whenever it was thought that the language could be made more explicit by changing a word, or even part of a sentence, this has been unhesitatingly done; and thus there are but few sections which have not undergone some modification; while, for the language of many and for the arrangement of the whole, the Committee must be held exclusively responsible.

Gross expressed the opinion that the code may not have been faultless, but it was as free from errors as it is possible for any code of morals to be. Hays was elected treasurer of the convention out of which grew the American Medical Association and was annually reelected from 1848 to 1852, when he declined being again a candidate for the office.

As chairman of the Special Committee on Representation at the sixth meeting in New York in 1853, Hays objected to the inequality in the ratio of representation and presented a remedy in the form of member to delegate ratio. He proposed that the Association be composed solely of members elected directly by it or through its Council and that this Council should be a permanent body, renewable in part each year, the duties of this Council to be the general superintending of the affairs and publications of the Association. These suggestions were not adopted. Later, by amending the law in regard to representation, the convention imperfectly accomplished the object aimed at by

Hays in the early sessions. Some of the early difficulties of the Association can be traced to the tardy acceptance of his ideas on representation. Members were elected on the basis of personal popularity and not on their professional merit or distinction. It seems probable that part of the disinterest shown in the Association for many years by the more cultivated members of the profession and the medical colleges was also due to this system of election. Many years elapsed before all of Hays' suggestions on the maintaining of separate sections for the discussion of general and specialized subjects and the creation of a Judicial Council to handle all questions of an ethical or judicial problem were carried out in full content.

Hays became a fellow of the College of Physicians of Philadelphia in 1835 and performed active work in its behalf. He was chairman of the building committee, took an especially active interest in the library and edited the transactions of the college for a number of years. In 1848 he was a delegate from the college to the convention for organizing the Medical Society for the State of Pennsylvania and the chairman of the committee which drafted the constitution for that society. The best way to summarize his organization work in behalf of medicine would be to restate that he was among the founders of more than a dozen associations, the oldest member of a dozen more and the chief officer of another dozen societies.

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MARGINAL DYSTROPHY OF THE CORNEA ASSOCIATED WITH PROLAPSE OF THE IRIS

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PHILADELPHIA

The ocular condition present in the patient whose case is to be reported has been described under various names, furrow keratitis, marginal degeneration of the cornea, ectatic marginal dystrophy of the cornea and other variants. Marginal dystrophy of the cornea seems most appropriate, as this feature is common in all cases and the term is in keeping with modern nomenclature.

The disease is rare. About 75 per cent of the reported cases were in males between the ages of 10 and 70 years. The condition is usually bilateral but is not always in the same stage of development in the two eyes. There is often a history of irritation of the eye and sometimes of persistent slight conjunctivitis. Rarely, rupture of the globe brings the first subjective symptom, or this complication may occur without subjective symptoms, as in the present case.

The furrow usually develops in the upper part of the cornea and may extend around the full circumference. It is preceded by opacity and peripheral vascularity. The opacity is in the form of a gray line. The furrow develops between this line and the margin of the cornea. The epithelium is intact. With the deepening of the groove the central edge becomes abrupt, and the floor either presents one or several localized ectatic areas or becomes ectatic in its entire extent as the result of the yielding of the thinned tissue to the intra-ocular tension. Seldom does ectasia occur in the inferior half of the cornea. The occurrence of ectasia is often delayed for many years. At several points the conjunctiva may be drawn over the area of ectasia in the form of pseudo-pterygia. In the area of the groove sensibility is absent. The intra-ocular tension is, as a rule, normal. Rupture of the floor of the groove, with resulting prolapse of the iris, usually follows slight trauma, though it may occur spontaneously, as is probably the case when the prolapse is bilateral. In Uhthoff's case, however, though the rupture was bilateral and occurred many years apart in the two eyes, it was attributed to a slight injury in both instances. In time the bulbar conjunctiva covers the protruding iris.

Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, April 16, 1936.

The alteration in the curve of the cornea diminishes visual acuity, and this is due in nearly all cases to the development of a high myopic astigmatism against the rule—in Ischreyt's case, 22 D. In several cases, including one recorded by Parsons, and in the case I reported in 1911, crystals have been present either in the uninvolved portion of the cornea or within the furrow. Handmann frequently observed degenerative changes in the macula.

Rupture of the cornea seems to be a rare complication. In Uhthoff's case it occurred in both eyes. In Schutz' patient, a boy aged 18 years, both eyes showed peripheral ectasia and in one eye rupture had occurred. Manes and Moulié recorded a case in a man, aged 37, in both of whose eyes there was prolapse of the iris. The herniated iris was covered with conjunctiva drawn onto the cornea.

Axenfeld and Gifford each performed a successful operation for the removal of cataract in an eye presenting marginal dystrophy. Both operators covered the incision with a conjunctival flap. Gifford first applied tri-chloroacetic acid.

The cause of the condition is unknown. According to Doggart, bacteriologic studies have thrown no light on the cause. Trachoma, iridocyclitis and glaucoma have been associated with the condition, and in one case there was a history of malnutrition. In both of Cattaneo's cases hypercholesteremia was shown, and Bohm and Fassel noted hepatic hypofunction in both their cases. Lugli suggested endocrine dysfunction.

In a case of Parson's, reported by Doggart, a study was made with the slit lamp. In the left eye endothelium was not visible in the middle portion of the ectatic zone, but its amorphous remains appeared in the lateral portion. Immediately below the two ends of the white line, the corneal stroma, for a distance of 1 mm., showed abundant glistening crystals and ill defined grayish opacities. In the lower portion of the cornea of this eye and the upper portion of the cornea of the right eye, which appeared normal on examination with the naked eye, conjunctival vessels encroached at various points for a distance of 1 mm., and a number of scattered grayish dots of opacity appeared in the substantia propria. No thinning of the cornea was yet demonstrable. These phenomena presumably denoted the beginning of the process the later stages of which were evident in the upper portion of the periphery of the cornea of the left eye.

The anatomic change which has constantly been noted is complete degeneration of the substantia propria and of Bowman's membrane. This is preceded by separation of their fibers and splitting of the lamellae of the cornea into loose fibrillar, richly nucleated tissue. The ends

of the fibers which reach the affected area are clubbed and wedge-shaped and are transparent. Fat globules are observed in the corneal lamellae in the vicinity of the furrow. These changes represent local degeneration of the corneal tissue and an effort to compensate for the loss of the corneal tissue by the formation of fibrillar tissue rich in nuclei and blood vessels. As this attempt is only partially successful, thinning of the diseased area of the cornea results, which manifests itself clinically in the formation of a furrow.

Instances of prolapse of the iris have been reported by Uhthoff (two cases), Seefelder (two cases), Schutz (one case) and Drüner and Wiedersheim (each one case). Fuchs, Seefelder, Rupprecht, Kur-yama and Coats have made histologic examination.

According to Gifford, who has reported several instances of marginal corneal dystrophy, the only others reported in the United States prior to 1924 were the cases of Knapp, Schutz, Gonzalez and Zent-mayer.

Doggart has completed the list of references to 1930. To these must be added the reports of Manes and Moulié,¹ Bohm and Fassell,² Lugli,³ Höhr Castán⁴ and Opin and Reboul.⁵

REPORT OF A CASE

On Oct. 21, 1935, Mrs. C. F., aged 52, was referred to me by Dr. M. E. Smukler. The patient was born in Russia and came to the United States fourteen years ago. There was a history of inflammation of the eyes during childhood. In 1930 she was treated for hyperthyroidism. The same year Dr. Smukler performed multiple ignipunctures for the relief of entropion and trichiasis of the lower lid of the left eye. Shortly before the patient was seen by Dr. Smukler, she had consulted a druggist because of a foreign body in the right eye. In removing the body he noted the prolapse of the iris.

Examination of the patient revealed trichiasis of the lower lid of the left eye and slight ptosis of the upper lid of the right. On both sides the sulcus of the superior lid was deep, so that a large part of the upper surface of the eyeball was covered by the lid. In both eyes there was marginal dystrophy of the entire circumference of the cornea. The resulting furrow was of uneven depth. In the right eye there was an area of prolapse of the iris about 3 mm. in diameter

1. Manes, A. J., and Moulié, H.: *Arch. de oftal. de Buenos Aires* 5:5 (Jan.) 1930.

2. Bohm and Fassell: *Sborn. oftal* 5:174, 1930.

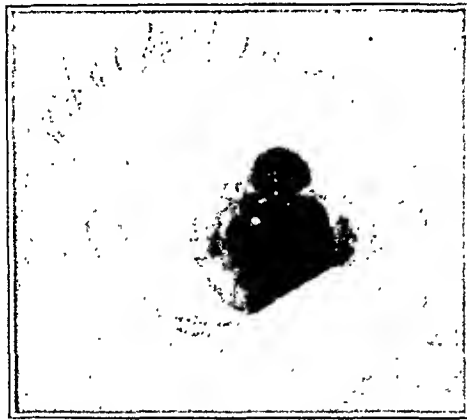
3. Lugli, L.: *Arch. di ottal.* 38:449 (July-Aug.) 1931.

4. Höhr Castán, J. M.: *Degeneración marginal de la córnea*, *Arch. de oftal. hispano-am.* 35:74 (Feb.) 1935.

5. Opin and Reboul: *Dystrophie marginale des cornées chez les jeunes sujets*, *Arch. d'opht.* 52:771 (Nov.) 1935.

through the floor of the furrow (figure). The protruding tissue of the iris was covered with epithelium. The sensibility of the cornea was subnormal. The epithelium was intact, as evidenced by the absence of staining with fluorescein. The tension in the right eye was 22 mm. and in the left 20 mm. A 1 per cent solution of physostigmine salicylate was instilled into the eye, with little or no effect on the size of the prolapsed area.

Biomicroscopic Examination.—As the corneal image of the light was swept across the limbus, it became constricted in the grooved portion. The constriction was particularly marked at the upper margin where a deep groove reached well into the substantia propria. The epithelium was intact. The vascular loops of the limbus extended farther into the cornea than normally. It was impossible to determine whether the endothelium was intact in the involved portion of the cornea, but it was normal in the surrounding area. No folds were visible in Descemet's membrane. There were disseminated areas of thinning of the uveal pigment in the zone of the sphincter. The lens was normal. The biomicroscopic observations were corroborated and elaborated by Dr. Alfred Cowan.



Photograph showing marginal atrophy of the cornea, with prolapse of the iris.

The error of refraction of the right eye was corrected by $-3.00 \text{ } \ominus +5.50$ cyl., axis 180° , with a vision of 6/9 part, and the error of the left, by $-0.75 \text{ } \ominus +4.50$ cyl., axis 90° , with a vision of 6/6 part.

Stated in terms of crossed cylinder equivalents, the correction for the right eye was -3.00 cyl., axis $90^\circ \text{ } \ominus +2.50$ cyl., axis 180° , and for the left eye, -0.75 cyl., axis $180^\circ \text{ } \ominus +3.75$ cyl., axis 90° . It is thus clear that in the right eye the myopic meridian was the higher and the astigmatism was against the rule, which is the usual finding in this condition, while in the left eye the hyperopic meridian was the higher and astigmatism was with the rule.

In 1911 I presented before a meeting of this section a case of this condition in a woman, aged 48. The condition was bilateral, with a well marked groove encircling the margin of the cornea except for an arc of about 15 degrees in the lower part of the inner portion. It lay peripheral to an area which was not uniformly dense and on which the furrow encroached in places. In several places small pseudopterygia and at other points yellowish dots, presumably hyaline granules, were present.

PRIMARY TUMOR OF THE OPTIC NERVE

REPORT OF THREE CASES

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Primary tumor of the optic nerve is not common, although in the literature of the past sixty-four years about 300 cases of tumor of the optic nerve have been reported. Four have been observed at the Massachusetts' Eye and Ear Infirmary in Boston in thirty-six years, and one, at the Wills Hospital in Philadelphia in twelve years. Hudson¹ found that this type of tumor was more common in females than in males and

led some authors to suggest that the neoplasm is congenital.

The tumor may spread from the intra-orbital to the intracranial portion of the optic nerve through the optic foramen or from the intracranial to the intra-orbital portion of the nerve. In possibly the majority of the cases reported the neoplasm has occurred in the intra-orbital portion of the nerve. An intra-orbital tumor of the optic nerve can be diagnosed with a fair amount of certainty. Usually the first symptom is a gradual monocular loss of vision in a young person (the patient being under 21, as a rule), followed by a painless proptosis of the globe on the affected side. The proptosis is in the orbital axis, and there is resistance when attempts are made to palpate the globe backward. Ocular movements are but slightly impaired. The fundus shows changes which are confined chiefly to the disk, such as papilledema or atrophy of the optic nerve. Of 118 cases reported by Hudson,¹ in only 3 was the fundus normal. In one half of the cases the fundus showed atrophy of the optic nerve, and in the other half it showed papilledema. Roentgenograms of the optic foramen are often of value as a diagnostic aid in these cases. The foramen may be enlarged considerably, as in case 1 of this series. Whitnall² gave the average diameter of the canal as 5.5 mm. for adults. The average for children is about from 4.5 to 5 mm.

From the Department of Ophthalmology, University Hospital, University of Michigan Medical School.

1. Hudson, A. C.: Primary Tumors of the Optic Nerve, Roy. London Ophth. Hosp. Rep. **18**:317 (July) 1912.

2. Whitnall, S. E.: Anatomy of the Human Orbit and Accessory Organs of Vision, New York, Oxford University Press, 1932.

The intracranial primary tumor of the optic nerve or chiasm cannot be diagnosed so easily. Martin and Cushing³ were able to diagnose such a tumor preoperatively in only 1 of 7 cases. There is usually no exophthalmos unless the intra-orbital portion of the nerve is involved. Loss of vision is slowly progressive in one or in both eyes. Observation of the visual fields is important and may show bitemporal or homonymous hemianopia or total blindness in one and hemianopia in the other eye. Roentgenograms may show deformation of the sella turcica and enlargement of the optic foramen. The fundus may show papilledema or atrophy in one or in both eyes.

The prognosis in these cases depends on the location of the tumor. If the intra-orbital portion of the nerve is involved and the tumor can be removed completely, prognosis for life is good. If the intracranial portion of the nerve is affected, the prognosis for life is poor, even though all the tumor can be removed. Martin and Cushing³ operated on 7 patients and only 1 survived. The possibility of recurrence is remote if all the tumor has been removed.⁴

The best method of treatment for such a tumor is surgical, although roentgenotherapy has been used with varying results in treating intracranial neoplasms when it was impossible to remove all the tumor. For the intracranial and for some intra-orbital tumors the cranial or frontal approach is best. This consists of removing an osteoplastic flap from the frontal region and elevating the frontal lobe of the brain. An intra-orbital tumor can be removed by the Krönlein method of resecting the lateral wall of the orbit.

REPORT OF CASES

CASE 1.—C. M., a girl aged 10 years, was admitted to the University Hospital on Nov. 13, 1934. For five weeks poor vision and a slight bulging forward of the left eye had been noted. The proptosis had gradually increased.

The results of physical examination were essentially negative.

Ophthalmologic examination gave the following results: Vision was 6/9 in the right and 6/30 in the left eye, and the tension to the fingers was normal in each eye. There was definite gross exophthalmos of the left eye; the protrusion as measured with the exophthalmometer amounted to 13 for the right and 18½ for the left eye. The eyes were straight and the extra-ocular movements normal. The conjunctiva were normal in both eyes; the pupils were equal in size and round, the left reacting more sluggishly to light than the right. The globe was displaced forward but in no other direction, and it could not be pushed backward. There was no tenderness; no abnormal orbital mass was felt, and no bruit was heard. Examination of the fundus of the right eye gave negative results; that of the left eye showed papilledema of from 3 to 4 diopters. No hemorrhages were seen. The veins were normal in the right and engorged and moderately tortuous

3. Martin, P., and Cushing, H.: Primary Gliomas of the Chiasm and Optic Nerves in Their Intracranial Portion, *Arch. Ophth.* **52**:209 (May) 1923.

4. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerves in Their Intracranial Portion, *Arch. Ophth.* **52**:209 (May) 1923.

in the left eye. The right visual field was normal; the left showed slight concentric contraction of form and color and enlargement of the blindspot.

Otologic and neurologic examinations gave negative results.

Roentgen examination showed minimal increase in the size of the left orbit and moderate enlargement of the left optic foramen.

Operation.—A diagnosis of tumor of the left optic nerve was made, and on November 24 the patient was operated on in the department of neurosurgery. A flap of the scalp was turned down in the left frontal region, an osteoplastic flap removed, the dura incised and the left frontal lobe elevated, exposing the intracranial portion of the left optic nerve, which appeared normal. The roof of the orbit was then removed, and the fatty tissue over the orbit bulged upward. When this was separated a large tumor, 15 mm. in diameter, almost globular, of bluish, almost translucent color was exposed. It appeared cystic, but aspiration revealed no fluid. There was only about 3 mm. of optic nerve between the eyeball and the mass, and this was enlarged. The optic nerve was divided close to the eyeball and the nerve found to be enlarged to the optic foramen. Therefore the foramen was opened and the nerve divided intracranially. The dura was then closed, the osteoplastic flap returned to its position and the incision closed.

Postoperative Course.—Recovery was uneventful. There were a definite pulsation and complete ophthalmoplegia with ptosis of the left eye. The disk was white, and there were hemorrhages about it. The veins were enlarged, and there were breaks in the blood column of the arteries. There were extensive pigmentary changes in the periphery of the retina. A few days later the fundus showed a more pink color. Two months later, on Feb. 4, 1935, the patient was in excellent health and had had no symptoms relative to the operation. The right eye was normal. The left eye showed a complete ptosis, with some function of the sixth nerve but none of the third and fourth nerves. Readings with the exophthalmometer were 16 for the left and 14 for the right eye. The fundus of the left eye showed a proliferation of vessels at the disk; the retinal vessels were replaced by white streaks, and the macula appeared grayish pink. In the periphery there were large areas of dense pigmentation and atrophy of the retina. When the patient was seen shortly before this report was made she was in good health.

Pathologic Diagnosis.—Examination of the tumor in the department of neuropathology revealed that the growth had invaded the perineural sheaths as well as the nerve itself. It consisted of unipolar and bipolar spongioblasts, forming a dense irregular and interwoven network. A diagnosis of spongioblastoma polare was made.

CASE 2.—R. A., a boy aged 6 years, was admitted to the University Hospital on Feb. 18, 1933. Two and one-half years before the child was admitted the mother noticed that the left eye was beginning to bulge outward. This bulging was more marked after the patient had had measles two years before he was seen. There was no complaint of blindness, but it was discovered that vision was defective in the left eye. The child had always been nervous. Recently he had complained of pain in the region of the forehead, especially in the morning. There had been no nausea or vomiting. The past history and the family history were irrelevant.

Physical examination and examinations of the blood and urine gave negative results.

Ophthalmologic examination showed vision to be 6/9 in the right and nil in the left eye. Tension to the fingers was normal in each eye. The pupillary reflex to light was lost in the left eye. There was definite exophthalmos, the protrusion as measured with the exophthalmometer amounting to 13 in the right and 18 in

the left eye. The left eye was proptosed in the orbital axis, and there was resistance to backward pressure. No mass could be palpated about the globe. In the fundus of the right eye the veins were slightly full but the appearance was otherwise normal. Examination of the fundus of the left eye showed that the media were clear, the disk was pale and had an infiltrated appearance and there was papilledema of 1 diopter. The veins were full and the arteries normal.

Roentgen examination showed definite alteration in the density of the posterior wall of the left orbit. There was also marked enlargement of the left optic foramen.

Neurologic examination gave no evidence of an intracranial lesion.

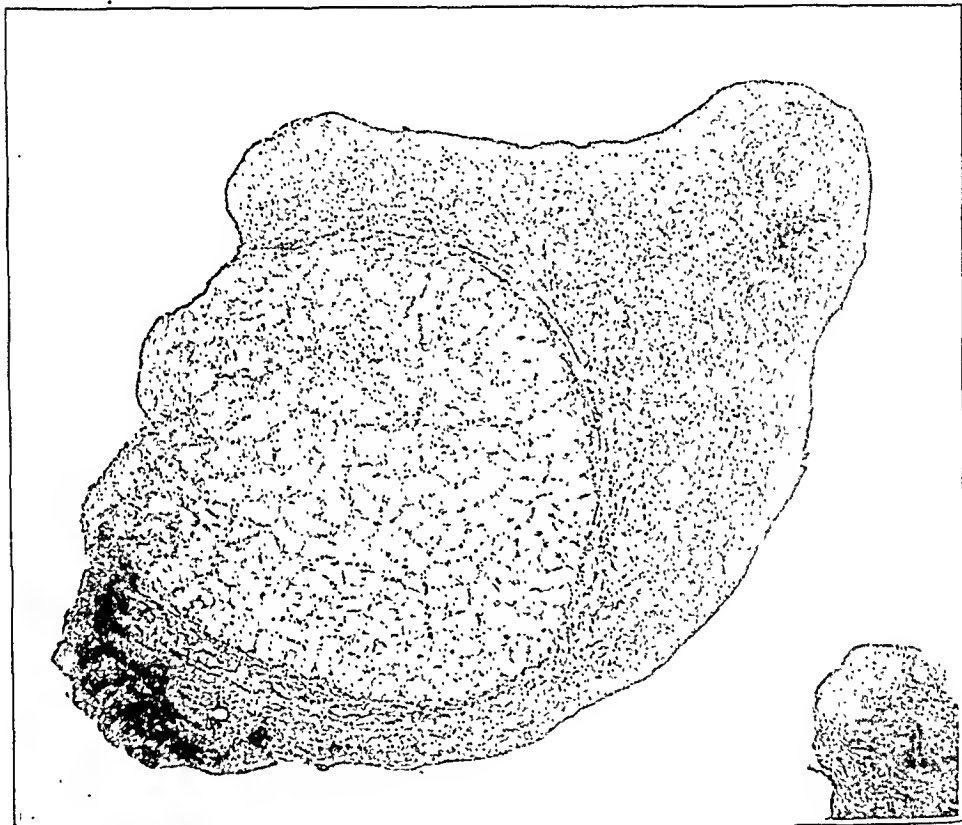


Fig. 1 (case 1).—Low power magnification of a cross-section of a spongioblastoma unipolare of the optic nerve, showing the tumor involving the nerve and perineural sheaths (azan stain, microplanar 35 mm.).

Operation.—A diagnosis of glioma of the left optic nerve was made, and on April 25 the patient was operated on in the department of neurosurgery. A flap of the scalp was turned down in the region of the frontal and parietal bones on the left, an osteoplastic flap removed and the dura incised. The left frontal lobe was elevated, a fibrous tumor of the left optic nerve being exposed. The tumor had passed through and posterior to the optic foramen; it was 1.5 cm. in diameter and of a bulbous appearance, being smaller toward the chiasm. It was believed to have extended into the left optic tract. The roof of the orbit was removed, and the tumor was found to extend to about 0.5 cm. from the globe. The nerve was divided on a level with the globe; the chiasm was divided close to the right

optic nerve, and the tract was divided back of the chiasm to get all the tumor tissue possible. The dura was closed, and the osteoplastic flap and flap of scalp were replaced.

Postoperative Course.—The convalescence was somewhat prolonged owing to low grade meningitis. This necessitated frequent lumbar punctures for several weeks. Five months later the patient's physician reported that vision in the right eye was nearly normal. Temporal hemianopia was present on the right because the left optic tract had been cut. The left eye was slightly lower in the orbit than the right. In the last report, on Sept. 11, 1934, the patient's physician stated that vision was normal in the right eye and that the patient was in good health.

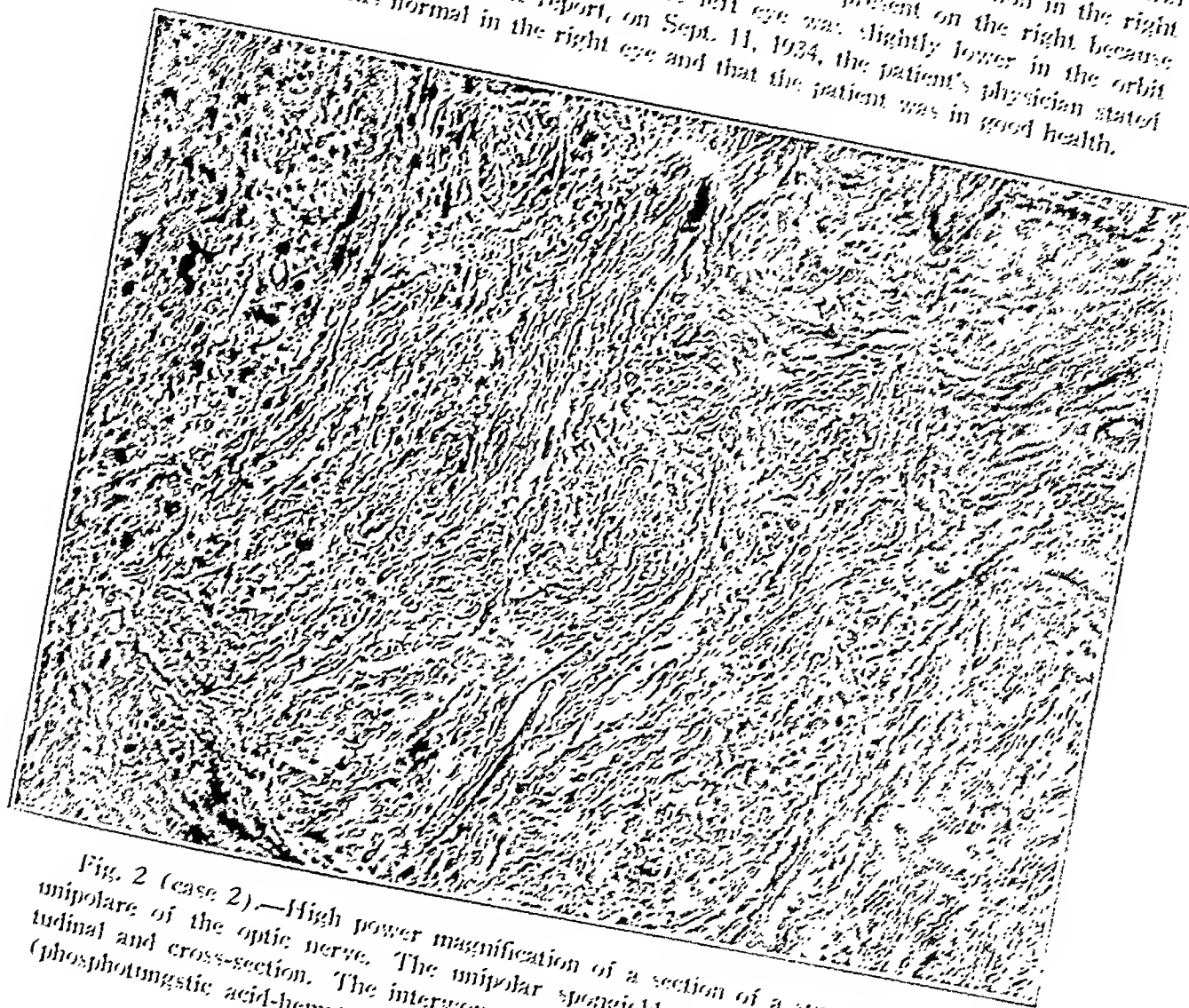


Fig. 2 (case 2).—High power magnification of a section of a spongioblastoma unipolare of the optic nerve. The unipolar spongioblast cells are cut in longitudinal and cross-section. The interwoven character of the tumor is clearly seen (phosphotungstic acid-hematoxylin stain, 0.16 mm.).

Pathologic Diagnosis.—The tumor was examined in the department of neuropathology. It was described as invading the optic nerve diffusely, bringing about its almost entire disintegration. The elements were unipolar spongioblasts in an early stage of development. There were still preserved bundles of nerve sheaths which were surrounded by a spongioblastic network frequently rich in fibers. The tumor tissue contained no glial tissue. There was no tendency toward necrosis. Lipoids were practically absent. A diagnosis of spongioblastoma unipolare was made.

CASE 3.—W. J., a boy aged 15, was admitted to the University Hospital on April 20, 1933. He complained of poor vision in the left eye, which had apparently not become worse since its onset six years before. There had been nausea and vomiting during the past two or three years, the vomiting being questionably of a projectile type. The patient was seen six years before by his home physician and given corrective glasses because of poor vision. The past history and the family history were irrelevant except for the fact that the patient had malaria at the age of 11 years.

Physical examination showed the patient to be well developed and not acutely ill.

Ophthalmologic examination showed the extra-ocular movements to be normal except for a constant, although irregular, vertical, oscillatory type of nystagmus. There was no deviation under cover, and the near point of convergence was normal. The conjunctiva, cornea, anterior chamber and iris were normal. The pupils were round and regular and reacted normally to light but sluggishly in accommodation. The fundi showed the media to be clear; the right disk showed diffuse atrophy with but little infiltration; the arteries and veins were normal; the maculae were

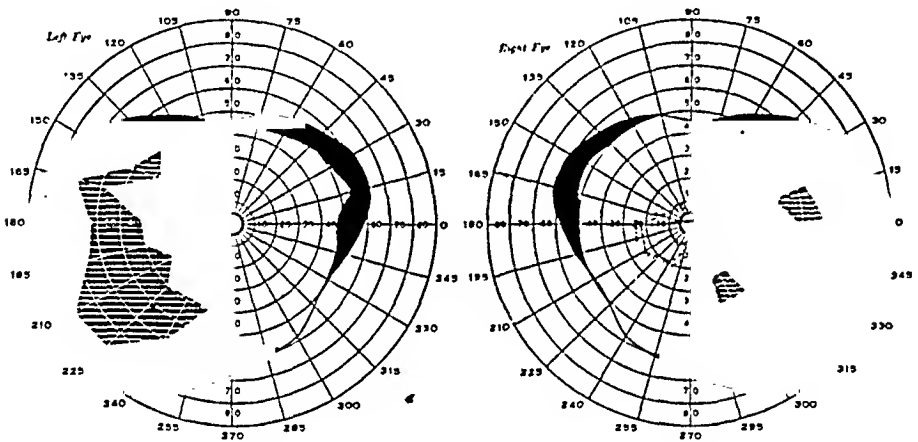


Fig. 3 (case 3).—Visual fields, showing bitemporal hemianopia with islands of vision in the temporal fields. The islands of vision are shown by the shaded areas. Vision was 6/6—in the right eye, and fingers were counted at 1 foot with the left. The test objects used were as follows: 5 mm. at 0.25 meter, 5 mm. at 1 meter and 10 mm. at 0.25 meter. In the right visual field the solid line indicates vision for green; the line of dashes, vision for red, and the dotted line, vision for blue. The colors were not seen with the left eye.

distinct, and no peripheral lesions were seen. The left disk was more atrophic than the right. Vision was 6/6—in the right eye; there was ability to count fingers at 1 foot (30 cm.) in the nasal field in the left eye. The visual fields showed bitemporal hemianopia with islands of vision in the temporal fields.

Roentgen examination was reported to show an increase in the prominence of the digital markings of the cranial vault and erosion of the sella turcica.

Operation.—A tentative diagnosis of pituitary tumor was made, and on April 21 an intracranial operation was performed in the department of neurosurgery. A flap of scalp and an osteoplastic flap were removed on the right side, the frontal and anterior part of the parietal region being exposed. When the dura was incised and the right frontal lobe elevated, a thin capsulated tumor about 35 mm. in diameter covering the sella turcica was exposed. The right optic nerve was

pressed slightly laterally, and the tumor arose from the left optic nerve which was entirely replaced with neoplastic tissue nearly up to from 1.5 to 2 mm. from the optic foramen. The nerve was divided at the optic foramen and a large portion of the tumor removed. The entire optic chiasm had been replaced by neoplastic tissue, but the right optic nerve showed no evidence of tumor, and hence was separated from the chiasm. The tumor extended back as far as the pons; hence no attempt was made to remove all the tumor. The dura was closed, and the osteoplastic flap and the flap of scalp were replaced.

Postoperative Course.—The patient did not recover consciousness and died twenty-four hours later. The temperature was 105 F. before death.

Pathologic Diagnosis.—The tumor was examined in the department of neuropathology and found to consist of elongated unipolar spongioblasts. The growth of connective tissue was moderate. A diagnosis of spongioblastoma unipolare was made.

COMMENT

The most common type of primary tumor of the optic nerve is the so-called glioma of subdural origin. Hudson¹ in 1912 stated that of 154 cases reported in the literature 118 were instances of glioma although only 28 were reported as such. In the past this type of tumor has been described as myxoma, fibroma, fibromyxoma, fibrosarcoma and myxosarcoma, probably because it was thought to be of mesodermal origin. Grinker⁵ and DeLong⁶ expressed the belief that all tumors of the optic nerve (gliomas) are of the spongioblastic unipolar or bipolar type, as in the 3 cases reported here.

Almost the only other type of tumor of the optic nerve is the endothelioma (if this can be considered, strictly speaking, a tumor of the optic nerve), which is of mesoblastic origin. The endothelioma originates in the nerve sheath surrounding the nerve and causes gradual atrophy. The differential diagnosis of glioma or spongioblastoma and endothelioma is not easy. In cases of spongioblastoma there is usually first blindness and then exophthalmos, while in cases of endothelioma the reverse is true. Restriction of ocular movements is more common in cases of endothelioma. The age of incidence in cases of endothelioma is greater, about 50 per cent of tumors of that kind occurring in patients over 30. In cases of endothelioma there may be circulatory obstruction shown in the lids and conjunctiva.

Neurofibroma of the orbit has been reported, but it is rare (4 cases are reported in the literature). The findings depend on the location of the tumor in the orbit and may easily be confused with signs of a tumor of the optic nerve. The neurofibroma is thought to arise from one of the various cranial nerves of the orbit. It commonly resembles a tumor

5. Grinker, R. R.: Tumors of the Optic Nerve, Arch. Ophth. 4:497 (Oct.) 1930.

6. DeLong, P.: Primary Tumors of Optic Nerve: Report of a Case, Am. J. Ophth. 17:797 (Sept.) 1934.

of the optic nerve when located deep in the orbit; when it is more shallow the globe is likely to be proptosed in an upward or downward direction, in contradistinction to straight forward.

SUMMARY

Primary tumor of the optic nerve is rare. It occurs more commonly in females, and 75 per cent of the patients with such a tumor are seen in the first decade of life. The tumor may be in the intra-orbital or in the intracranial portion of the nerve or in both. If it is in the intra-orbital portion it is more easily diagnosed. The symptoms are a gradual monocular loss of vision, usually a painless proptosis of the globe in the orbital axis and papilledema or atrophy of the optic nerve. A roentgenogram often shows an enlarged optic foramen. If the tumor is in the intracranial portion of the optic nerve there is a gradual loss of vision in one eye or in both eyes, without proptosis. The visual fields show bitemporal or homonymous hemianopia or total blindness in one eye and hemianopia in the other. A roentgenogram may show deformation of the sella turcica. The prognosis in cases of intra-orbital tumor is fairly good if the tumor can be removed, but in cases of intracranial tumor it is poor because of operative risk and extension of the tumor to vital parts of the brain. In the 3 cases reported here the tumor was of the spongioblastoma unipolare type.⁷

7. Permission was granted by Dr. Max Minor Peet, of the department of neurosurgery, for the use of the operative report in each case and by Dr. Konstantin Lowenberg, of the department of neuropathology, for the use of the pathologic description in each case.

Clinical Notes

AN IMPROVED PERIMETER-CAMPIMETER

JOSEPH I. PASCAL, M.D. NEW YORK

A combination perimeter and campimeter is described which is designed for every-day clinical work. It takes a middle course between the very elaborate instruments for laboratory use and the very simple ones intended only for use in obtaining approximate results. It embodies several novel features which I believe help to make field testing more rapid and less fatiguing to both doctor and patient.

DESCRIPTION OF THE INSTRUMENT

The arc of the perimeter, 9.2 cm., is sufficiently broad to supply an adequate background for both fixed and moving targets. The radius of the arc is 315 mm., which is a convenient working distance and yet long enough for good clinical work.

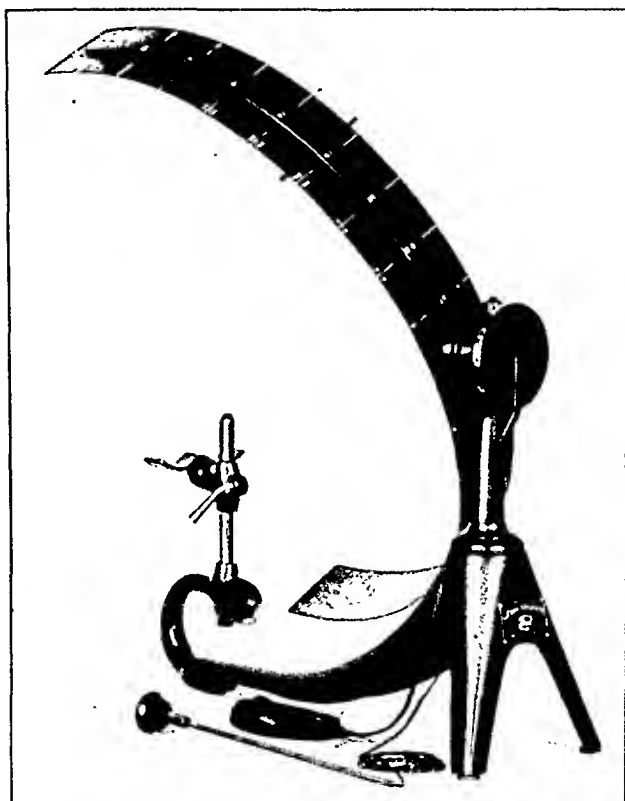
Fixation Target.—The instrument aims to make fixation steadier and more restful by supplying a variety of fixation targets. These can be interchanged by the turn of a finger, and thus several fixation targets can be provided in order to hold the patient's attention. The mechanism for this purpose consists of a small 10 mm. plane mirror set in the middle of the perimetric arc. Below the patient's chin, attached to the supporting bar, is a small housing carrying a small 10 cent electric bulb connected by means of a rheostat to the general current. In front of the bulb are two revolving disks. One disk contains an occluder and five different apertures. These are a 10 mm. round, a 5 mm. round, a cross, a triangle and a clear ring with a central black dot. The latter provides a "negative" fixation target. The other disk carries five colored patches, white, blue, yellow, red and green, each one of which can be exposed behind any one of the openings of the first disk. The patient looks into the mirror and sees the reflected fixation target. The mirror acts like a long tube and insures steady fixation, since only when the patient fixes accurately is the target visible. The targets may be changed in size, form, color or intensity by a turn of the finger. The occasional changing of the form or color of the fixation target serves to hold the patient's attention on the target much longer, and fixation then proves less fatiguing to the patient and the examiner alike.

An additional factor which insures steadiness of fixation is the fact that the fixation target is farther from the examined eye than the movable target. The reflected fixation target is about 70 cm. from the eye, while the movable target is at 31.5 cm. from the eye. It is a fact easily demonstrable that when the central fixation target is beyond the arc of the perimeter and farther than the moving peripheral target there is less tendency on the part of the patient to turn his eye from the fixation target toward the moving target.

This paper was read before the Section of Ophthalmology, New York Academy of Medicine, on March 16, 1936.

Central Color Scotomas.—Central relative scotomas can be easily detected by revolving the color disk. This brings each of the four principal colors directly into the central field without first exposing it in the paracentral field.

Moving Target.—The moving target is set in a small housing mounted on a long black handle. Here likewise there are two superimposed disks, and by a turn of the finger targets of different size and color can be presented. The sizes chosen are 1, 2, 3 and 5 mm. In addition to white, the four principal colors can be presented in these different sizes. This enables the examiner to do quantitative perimetry as well as quantitative color perimetry or a combination of the two. The targets are made self-luminous by means of a small ordinary 10 cent bulb set in the housing. This arrangement provides even illumination in all parts of



An improved perimeter-campimeter

the field. There is no diffuse illumination, but the brightness of the target can be altered to suit various conditions. Perimetry can be carried on with equal ease in a light room and in a dark room with a dark-adapted eye. For very fine work at the margin of the field instantaneous exposure can be had by means of a switch in the handle which flashes the current on and off.

Accessory Degree Numbers.—The degree numbers are shown by a scale at the back of the perimeter arc and also by a series of small perforations at the rim of the arc. This enables the examiner to see the position of the moving target from every direction and for every position of the arc. Whether the examiner works alongside, in front of, or behind the patient he can at all times see the perforated degree numbers without having to bend round the arc in order to see the scale on the back.

Campimeter.—For a study of the physiologic blindspot and of central and paracentral scotomas, a flat disk campimeter is provided. This is readily attached to the perimeter arc without disturbing or moving the patient. Thus a perimetric and campimetric examination can be carried out with a minimum of time and energy. This disk is set 25 cm. from the eye. At this distance defects of the central and paracentral field can be conveniently mapped, and yet the distance is long enough to bring out even small defects. The mapping can be made on the slate and transferred to a record chart, or a chart the duplicate of the slate may be inserted in the campimeter frame and the record filed directly.

Since writing this article my attention has been called to a description of an improved perimeter and screen by Lewkowitsch¹ of London, England. There are several features in the two instruments which are more or less similar as well as several in each of the instruments which are entirely different and unique.

1. Lewkowitsch, H.: An Improved Perimeter and Screen for Examining the Field of Vision, Brit. J. Ophth. 5:166 (April) 1921.

Ophthalmologic Review

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FIXATION IN SCOTOMETRY

A CRITICAL INQUIRY

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The ability of the eye to fix a point of reference is essential to standard methods of scotometry. Though fixation theoretically may be accomplished by any portion of the retina, it is certainly necessary that the visual line remain (relatively or absolutely) stationary in order to permit the plotting of a defect.¹ Fixation of a point of reference on a chart by the macula constitutes the standard by which the anomalous types of fixation are judged, but macular fixation is in itself a complicated process, and many of the elements concerned are as yet poorly understood. In the following pages an effort is made to summarize certain aspects of the problem so as to emphasize the importance of this act in scotometry.

If a defect is projected on a tangent screen placed at 2,000 mm. and again on one at 330 mm., it is found by calculation that the first screen gives an enlargement of the defect that is about six times greater than that on the second screen. One would therefore think that it would be much easier to outline the defect accurately at 2,000 mm. This is doubtless so in a gross sense, but the details are lost, and in the case of a scotoma the detection of details is a most important essential. Why should detail be lost in this instance with magnification? There are perhaps a number of reasons, but the most obvious seems to lie in the fact that fixation can be maintained most perfectly at shorter ranges.

It is necessary to inquire into the mechanism of fixation in order to comprehend just what this means. Since the macula is the "organ" of fixation, its functions must be catered to. Therefore, macular function is the first step to be considered in this inquiry.

From the Department of Ophthalmology, the Long Island College of Medicine.

1. Prism scotometry and scotometry in which the scotoma is caused to pass over a fixed point by directed movements of the visual line are omitted from this consideration.

MACULAR FUNCTION

The fixation spot must have a high degree of visibility (as the macula is most efficient for objects of this sort). Factors which make for visibility are (1) a high degree of reflection, (2) great amount of contrast and (3) refinement of detail. The amount of light falling on the fixation point does not of itself imply good visibility, even if the contrast between the fixation point and the background is good. The amount of light reflected back to the macula is of vital importance. In this regard, it is necessary to recall certain physiologic peculiarities of the macula, however. First is that truism stated by Helmholtz:² "The dimmer the light, the farther away [from the fovea] is the part of the retina that is used in looking at it." Moreover, when the fixation point is poorly illuminated, the degree of eccentric fixation is constantly changing on account of retinal adaption to the new conditions.³ This is not an assumption without material support. The following statement is quoted from Parsons:⁴ "The rate of regeneration of the substance is quicker for the cones than for the rods, which accounts for the much quicker adaption of the former (cones 3 minutes; rods 30 minutes)." The change from the mechanism of the rods to that of the cones occurs at a brightness of 0.0134 millilambert.⁵ Hecht calculated that there are 572 steps in the discrimination of luminosity, and that one-third are due to rod and two-thirds to cone vision. This theory accounts for the falling off of the ability to discriminate changes in luminosity at both high and low intensities.⁶

A fixation spot loses brightness inversely as the square of the fixation distance; hence it loses much of its brightness when moved from 190 to 2,000 mm.⁷ (over one-hundredth as much light). This great reduction may be so marked as to be appropriate for fixation by an eccentric

2. Helmholtz, H.: *Helmholtz's Treatise on Physiological Optics*, translated and edited by J. P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1924, vol. 2, p. 332.

3. Helmholtz,² p. 333.

4. Parsons, J. H.: *An Introduction to the Theory of Perception*, New York, The Macmillan Company, 1927, p. 228.

5. Luckiesh, M., and Moss, E. E.: *Seeing*, Baltimore, Williams & Wilkins Company, 1931, p. 63. The authors discussed the relationships of various fundamental and practical units involved in brightness. Perfect diffusion is assumed when necessary in accordance with the definitions.

6. Duke-Elder, W. S.: *Textbook of Ophthalmology*, London, Henry Kimpton, 1932, vol. 1, p. 897. Duke-Elder in referring to low illuminations said: "In these conditions the paracentral region is the most sensitive, being almost 1000 times more so than the fovea."

7. Comparing the stereocampimeter range with that of the tangent screen at 2 meters.

retina, which is better adapted for vision in dim light.⁶ An eccentric retina, as already stated, adapts for form more slowly than the macula, so that "wandering" movements are set up, though of small amplitude.⁸ These can be demonstrated by the study of after-images projected on a screen at 2,000 mm.

That the fixation point must stand out against the background with a maximum amount of contrast is obvious if maximum brightness (relative) and detail of form are to be attained. Maximum contrast is attained when a background is used which absorbs the greatest possible amount of incident light and when a fixation point is used which diffusely reflects⁹ the greatest possible amount of incident light. A black background and a white point are hence to be sought, but one must keep in mind other factors having to do with the problem of general illumination.

The spot must have detail of form, as the macula is adapted to the recognition of form. The size of the fixation spot should be relatively minute, in fact approaching the threshold of the particular macular vision of the eye concerned.¹⁰

One may exaggerate the conditions again to make more emphatic the points discussed. The details of form of a fixation object are greatly reduced also when moved from 190 to 2,000 mm., as the size of the fixation object is not ordinarily increased when the screen is moved farther away. Thus, the ideal fixation object might be a letter from a printed page; it should probably subtend a visual angle corresponding to the subject's central (or, where this is reduced, paracentral) visual acuity, taken under the same lighting conditions as those in which the studies of the fields are made (from 7 to 20 foot candles). This would have form and would be appropriate for the macula to fix on at 190 mm., but when moved to 2,000 mm. it would become only a blurred spot.¹¹

If one were to use a round button or pinhead, that principle would still pertain, for its sharpness of border, its contour and its reflexes might be recognized as giving it form at 190 or 330 mm., but it would

8. Contrast is not disturbed, as the reduction of light on the background and on the fixation point is relatively unaltered. One must keep in mind, however, that Weber's law does not hold either for very high or for very low levels (Parsons,⁴ p. 186).

9. This is called diffuse reflection in contradistinction to specular reflection. The latter condition must be carefully avoided or glare results.

10. Helmholtz,² p. 301.

11. In actual practice this condition is inserted when studies are made on the tangent screen at 1 meter and then at 2 meters. One does not actually have occasion to move a screen from 190 to 2,000 mm.

be seen as a white blur at 2,000 mm.¹² This lack of form would render it appropriate for eccentric fixation but not for macular fixation.

The color of the fixation point probably should be white in order to enhance the contrast. The ideal fixation point would thus appear to be white, showing minute detail. On the other hand, if a dull, rather large oval ellipse were used, that isopter (of visual acuity) zone of the eccentric retina to which it corresponded would adapt for it, so that one could say that the eccentric retina was used to maintain the eye in the position of normal fixation. One concludes, then, that a chalky white letter of the alphabet, 5 minutes of arc in size, placed on a lusterless background (chart) would be an appropriate fixation point if macular function were intact.

In opposition to the ideas of a bright fixation point for the macula, it has been suggested that a bright fixation point is likely to fatigue¹³ the retina easily, with the result that an after-image is formed. The retina, not yet adapted, is then shifted to one side in order to replace the image. (However, retinal fatigue, in this sense, probably does not occur.) Such a movement is of course incompatible with refined mapping.

Whether stereoscopic (binocular) fixation is used or not, it is possible to combine the central and eccentric zones for fixation by arranging lines on a chart in such a way as to give the effect of looking down a tube. An objection to this perspective appearance of the fixation spot is that it stimulates the accommodative effort. This aspect will be considered next.

INFLUENCE OF ACCOMMODATION ON FIXATION

Since recognition of the detail of near objects is normally dependent on accommodation and since satisfactory fixation depends on the maintenance of a sharp definition, it is obvious that accommodation bears an important relation to fixation. Near vision for the recognition of detail is a provision of nature.

Helmholtz,¹⁴ in discussing the form sense, recalled a significant fact:

The resolving power is much less in the lateral parts of the retina than in the yellow spot; the decrease near the center of the retina being less than it is farther away from it. . . . Another noteworthy result from these measurements is that

12. To be sure, under ordinary conditions (when the eye is adjusted for infinity), the blur circle for 330 mm. is about 0.1 mm. larger than one for a distance of 2,000 mm. (Helmholtz,² vol. 1, p. 132), but it will be shown in the following paragraphs how the blur circle for near vision may be reduced to the proportions of one at infinity.

13. There is a growing tendency to oppose the idea of retinal fatigue (Duke-Elder,⁶ p. 968; Parsons,⁴ p. 180).

14. Helmholtz,² p. 34.

in accommodation for distance, the falling off of the resolving power toward the periphery of the retina seems to proceed more rapidly than it does in near vision.

Tscherning¹⁵ gave definite data on this point.

Prolonged maintenance of steady accommodation while a nearby stationary object is being fixed seems to be an unusual combination, irrespective of the distance of fixation. If the accommodation can be relaxed entirely, fixation seems easier; in fact, when one is attempting to fix a point on a tangent screen, one will often become conscious that the eye is entirely out of focus. (While this may be detrimental to accurate fixation, it does not interfere with reasonably accurate plotting of eccentrically located defects.) I have had a +10 diopter lens made with the center (2 mm. in area) ground plane. As fixation through the center is sharp, eccentrically located defects are as readily plottable (though slightly enlarged) as if no such lens were before the eye. This type of lens steadies fixation, because if the subject attempts to look at the object directly, he is at once reminded of the act by the blurring of the +10 diopter part of the lens. The ideal condition seems to be to fix the point clearly at the reading distance yet not to exert the faculty of accommodation.¹⁶ Fortunately this can be accomplished fairly easily by the use of the appropriate collimating lens (fig. 1).

Light reflected from a fixation point follows a divergent course. If the screen is placed 2,000 mm. from the subject, these rays can be rendered parallel before they enter the patient's eye by placing a +0.50 diopter lens before the eye.¹⁷ As it is unnecessary for the eye to accommodate for parallel rays, the fixation point may be seen without this exertion. A +1.00 diopter lens would be placed in the same way for a screen at 1,000 mm.; a +3.25 diopter lens, at 330 mm., and a +5.25 diopter lens, at 190 mm. The screen is thus placed optically at infinity, but it is actually at accommodative close range. The details are, moreover, slightly enlarged (at first) by this collimating lens, and the subject seems to be able to respond more definitely when the object disappears and reappears at the borders of a defect. The sharper details being more appropriate to the macular functions, fixa-

15. Tscherning, M.: *Physiological Optics*, ed. 4, translated by C. Weiland, Philadelphia, Keystone Publishing Co., 1924, p. 209.

16. Nagel (in Zoethout, W. D.: *Physiological Optics*, Chicago, Professional Press, 1927, p. 152) made this interesting statement: "With characters of the same apparent size, small characters nearer the eye were easier to discover than larger characters farther away." He said that the reason for this is difficult to explain and suggested the possible influence of accommodation.

17. Rays of light arising from a point 1 meter away are rendered parallel by a +1 diopter lens. A point at half this distance would require a +2 diopter lens to render the rays parallel. It is thus simple to calculate the lens which will collimate the divergent rays from a chart at any desired distance.

tion is encouraged. When the screen is placed at 190 mm. (as in the stereocampimeter), there is linear magnification (by the collimating lens) of about 15 per cent.¹⁸

When a scotoma is successfully plotted at different ranges, not only is there an increase in the size of the defect, but the defect becomes "thinner." If a defect is plotted at 6 meters instead of at 2 meters, it is more easily appreciated. The borders are less definite, on account of the increased influence of poor fixation, but the retina projected on the infinitely removed sphere of the visual field (at 6 meters) is spread over so great an area that patients often describe haziness for far vision but absence of objects for near vision (within the area of the scotoma). Hence this increase in the loss of definition of a defect shows a relation to the distance of fixation. If this same principle is carried further, it is found that a bit of black paper pasted on the lens of an eyeglass produces only a relative scotoma, and the same is true of corneal

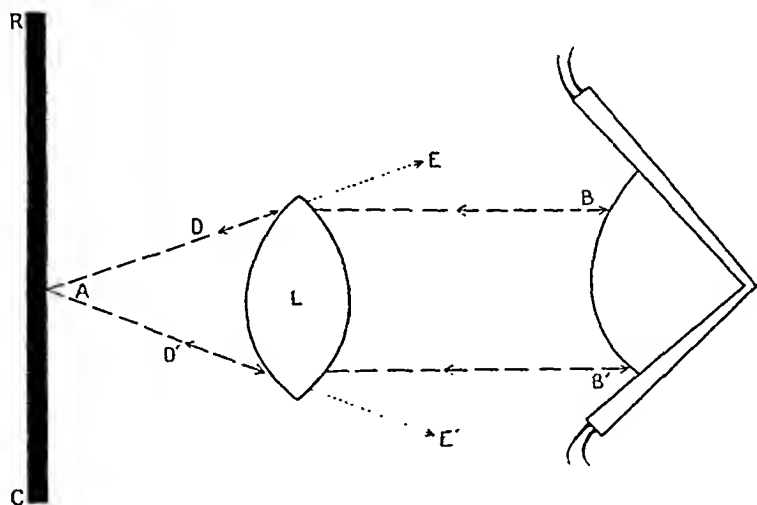


Fig. 1.—The collimating lens. *RC* represents the tangent screen and *A* the fixation point. Lines *ADE* and *AD'E'* show divergent course of light from the surface of the tangent screen. Divergent rays *AD* and *AD'*, impinging on lens *L*, are rendered parallel (*ADB* and *AD'B'*), so that they enter the eye as if from infinity.

opacities unless very minute test objects are used. Vitreous opacities, however, become so positive as to appear densely black to the patient.

The collimating lens has, in effect, the power of contracting the visual field, thus compressing, as it were, the relative defects into positive ones.

18. This figure is obtained by placing a -5.25 diopter lens over the campimeter lens of one side, thus neutralizing the $+5.25$ diopter lens in the instrument. A green glass is placed before the same eye. The prisms of the instrument are then adjusted until the green-appearing chart overlaps the opposite chart and the difference in size can be measured by counting the degree squares (smaller squares). (The central partition on my stereocampimeter is removable.) It is found that 20 degrees of the larger are occupied by 17 degrees of the smaller (Green).

MUSCULODYNAMICS OF FIXATION¹⁹

It is obvious that the anatomic position of rest would not be appropriate for fixation, because in total ophthalmoplegia externa and immediately after death, when the anatomic position of rest may be said to occur, the eyes rarely look straight ahead but usually diverge or occasionally converge. Should such a position be desirable, there is no known way of maintaining it. The extra-ocular muscles maintain a normal tonus at all times. When the eyes are uninfluenced by stimuli for orientation (accommodation, muscle balance, etc.) they assume what may be called the physiologic position of rest. It represents a balance of muscle tonus of the extra-ocular muscles. Such a state of affairs occurs only when both eyes are looking at the same point in infinity, and this occasion must arise rarely, unless artificial conditions are created to accomplish it (as with the stereocampimeter). This position, even when it does occur, being dependent on a perfect balance of muscular interaction, theoretically could not be stationary, because of the inherent irregularities of muscle contraction²⁰ during the maintenance of normal tonus.

Fixation, as it is commonly known, particularly for points inside infinity, has the result of an unequal distribution of work among the extra-ocular muscles, so that one has the right to expect the insertion of other factors further disturbing the theoretically perfect fixation. The actual movements of the eye during fixation have been studied elaborately by many workers and by many methods, and there is a surprising unanimity in the conclusions. It seems to be agreed that the efforts of the extra-ocular muscles to maintain perfect fixation results in minute jerky movements of the eyes,²¹ which occur in two components:

1. Relatively large jerky movements through an angle of 4 minutes of arc occur at intervals of from one to two and one-half seconds (fig. 2).

19. (a) Duke-Elder,⁶ p. 579. (b) Adler, F. H., and Fliegelman, M.: Influence of Fixation on the Visual Acuity, *Arch. Ophth.* **12**:475 (Oct.) 1934. I use Duke-Elder's figures in the following discussion because he apparently drew his conclusions from the reports of a great many workers. This seems safer than to accept the data of a single worker.

20. The simplest evidence of this phenomenon is the muscle sounds, which are easily elicited if an appropriately formed stethoscope is applied over the "belly of the extra-ocular muscles."

21. While it is not possible to cite actual evidence, it seems obvious that binocular fixation may modify the fixation movements to such an extent as to warrant the statement that monocular fixation is to all intents and purposes initiated by an entirely different mechanism than is binocular fixation.

2. During the period of rest there are fine jerky movements which have an amplitude of 1 minute of arc and which last for from one to two and one-half seconds. There is also a minute variation in the position of the head.

Adler and Fliegelman^{19b} concluded from a carefully controlled series of studies that there are three components: (1) rapid shifts occurring once every second, with an amplitude of 30 minutes of arc; (2) waves, occurring once every two seconds, with an amplitude of 3 minutes and

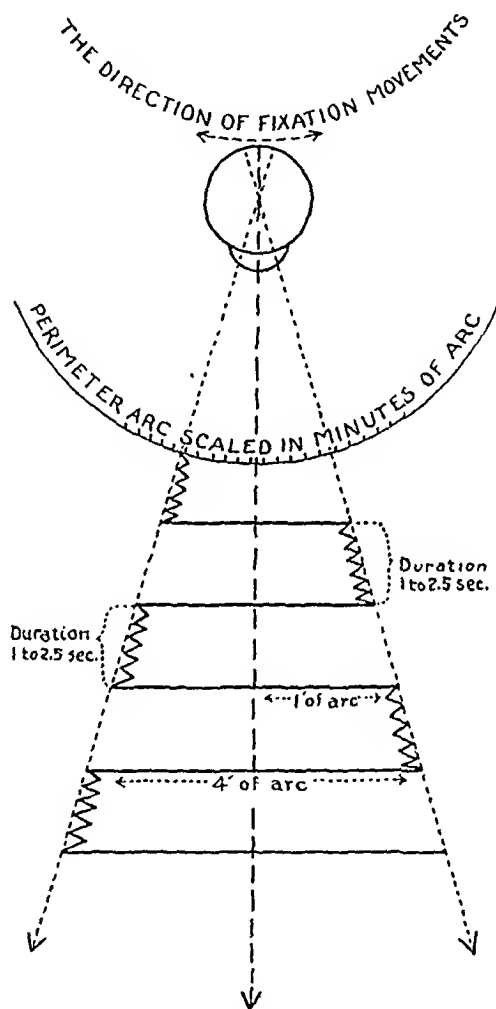


Fig. 2.—Schematic representation of the movements of the eye during fixation.

1 lambert = 0.3183 candle per square centimeter = 2.054 candles per square inch.

1 candle per square centimeter = 3.1416 lamberts.

1 candle per square inch = 0.4868 lambert = 486.8 millilamberts.

1 millilambert = 0.001 lambert.

1 foot lambert = 1.076 millilamberts.

1 spherical candle emits 12.57 lumens.

1 foot candle = 1 lumen incident per square foot.

1 lux = 1 lumen incident per square meter.

1 lambert = 1 lumen emitted per square centimeter.

1 lumen emitted per square foot = 1.076 millilamberts.

1 foot lambert = 1 lumen emitted per square foot.

1 millilambert = 0.929 lumen emitted per square foot.

43.5 seconds of arc, and (3) fine vibratory movements lasting for one one-hundredth or two one-hundredths second, with an amplitude of 2 minutes and 14 seconds of arc. In other words, once every second the eye makes a readjustment which carries the image over twenty-six cones. Furthermore, when the eye is steadiest, a point image is moving across the retina an angular distance of over 2 minutes, and even then it maintains one position for not longer than one-fifth second.

This means that the actual area of retina used for fixation is constantly shifted about over a relatively large arc. The amplitude of these movements is apparently the same whether fixation is for far or for near vision. They bear no relation to the pulse or the respiratory rate, and there is marked individual variation in the rate and duration. The movement is in general horizontal, but there is a rotary element in all probability so that the path of a retinal point will be an irregular ellipse.

Besides these fixation movements, there are movements associated with winking: (1) recession of the globe of about 1 mm.²² and (2) upward turning of the globe of about 15 degrees.²³ An explanation of the origin of these movements is not of interest here, as it is of no concern whether they occur as a part of the mechanism for the maintenance of retinal activity in the area of image formation or whether they arise entirely within the neuromuscular mechanism without the influence of sensory stimuli. Graphically, these movements may be depicted as in the accompanying illustration (fig. 2).²⁴

It is obvious that such movements result in the passage of an image over a minute area of the retina, for instance, with the movement of 4 minutes of arc²⁵ the image passes over about 0.02 mm. of retina (the movement of 1 minute of arc covers about 0.005 mm. on the retina). If a long pointer could be attached to the visual line so that it would move with these oscillations of the globe, it would traverse an appreciable distance on the tangent surface instead of remaining steadily in one place.²⁶ Calculations to show the extent of this area from the

22. Duke-Elder,⁶ p. 581.

23. Miles, W. R.: Elevation of the Eyeballs on Winking, *J. Exper. Psychol.* 14:311 (Aug.) 1931.

24. Luckiesh, M.: *Seeing and Human Welfare*, Baltimore, Williams & Wilkins, 1934, p. 96. The author said: "The average stopping-time of the eye is .17 second. In extensive tests of this it was found that the briefest interval of time that it is possible for an observer to fixate an object in order to gain an adequate visual impression varied for the most part from .07 to .30 second. The average was .17 second."

25. Taking the distance from the second nodal point to the retina as 17.055 mm. and multiplying it by the natural tangent value for 4 minutes of arc (or for 1 minute of arc).

26. The natural tangent value for 4 minutes of arc is 0.001164 mm.; for 1 minute it is 0.000291 mm.

various distances of fixation, as met with in standard instruments, give the following results:²⁷

Coarse Movement of Fixation		
Distance of Fixation, Mm.	Minutes of Arc	Tangent Plane, Distance in Mm.
190	4	0.22116
330	4	0.38412
750	4	0.87300
1,000	4	1.16400
2,000	4	2.32800
Fine Movement of Fixation		
190	1	0.05529
330	1	0.09603
750	1	0.21825
1,000	1	0.29100
2,000	1	0.58200

In other words, if a pointer could be attached to the eye, 4 minute movements would cause it to oscillate on a tangent screen placed at a distance of 2,000 mm., a distance of 2.328 mm.; at 1,000 mm., 1.164 mm.; at 330 mm., 0.384 mm.; at 190 mm., 0.221 mm. It thus becomes obvious that a minute scotoma as projected by the retinal defect is not stationary on the tangent screen and that the more remote the screen, the greater the area covered by the shiftings. The borders are thus much more difficult to plot and in fact may be missed entirely unless all other conditions are particularly favorable.

From these figures it might seem impossible to discover a scotoma as small as one equal to 4 minutes of arc, because the movements of the eye would carry it rapidly back and forth and so bring the image of the object on a seeing portion of the retina. This is not actually so, for it is easy to detect a scotoma which has reduced vision from 20/20 to 20/30. Such a defect can apparently be appreciated during the fine fixation movements, however.²⁸ In spite of these movements, Luckiesh,²⁴ has demonstrated that the eye can gain adequate visual impressions by fixating for 0.17 second; yet the finer movements are going on during that time.

Added to the actual movements, however, are other factors which, theoretically, distort the scotoma. These are retinal light scatter,

27. It must be realized that these figures represent the projection of minutes of arc on a tangent surface.

28. If the shape of the letters cannot be distinguished in the 20/20 line but can be in the line of larger letters, there is a defect in retinal function equal to the difference in these two letters. The diameter of the limb of a letter in the 20/20 line is 1.77 mm., while that of one in the 20/30 line is 2.65 mm.; hence the difference is 0.88 mm. The fine fixation movements (at 6 meters) cover 0.29 mm.; hence they would occur entirely within the defect. The coarse movements cover 1.16 mm.

spherical and chromatic aberration of the refractive media, other optical defects of the eye and probably minute discrepancies of the conducting and registering neuromechanism. These factors make it evident that the geometric relations of the functionally depressed retina and those of the projected defect (scotoma) cannot be stated in precise mathematical terms. The average width of the angioscotoma near the blind-spot of Mariotte is 1 degree. If this is projected at various distances on the tangent screen it is found that at 2,000 mm. it is 34.9 mm. wide; at 1,000 mm., 17.4 mm. wide; at 330 mm., 5.7 mm. wide; at 190 mm., 3.3 mm. wide.

When attempts are made to map a scotoma at 1,000 or 2,000 mm. ranges and the examiner finds that the object seems to fade from view in relatively large areas, he must realize that this is due not to "retinal fatigue" but to the indefiniteness of the border of a scotoma from 18 to 35 mm. wide. There is no sharp border to such shadows; they have a sort of penumbra.²⁹ Moreover, one would not expect a retinal lesion of measurable proportions to result in the production of a scotoma of predictable relative size, at least in a geometric sense. The fact remains that, in spite of these discrepancies, minute defects can be mapped, just as small print can be read, and one may be justified in assuming that this is possible by means of a sort of scanning, like search-lights sweeping across the night skies in search of an airplane. The recognition of the border of a scotoma, then, is akin to the recognition of the alinement of two hairs; the recognition is the result of the averaging of a group of stimuli rather than the product of a single fleeting impression.³⁰

29. Bailliart, P.: *The Retinal Circulation in the Normal and Pathological States*, translated by J. E. Lebensohn, Chicago, Professional Press, Inc., 1928, p. 16. Bailliart said that the retinal arteries as they emerge from the disk are 210 microns (0.21 mm.) in diameter and the veins are 245 microns (0.245 mm.) in diameter.

30. The following quotation (Adler, F. H.: *Clinical Physiology of the Eye*, New York, The Macmillan Company, 1933, p. 136) will serve to emphasize this point: "These movements must not be looked upon as defects which the individual tries to overcome in order to see acutely. As a matter of fact, if absolute fixation could be obtained it would be a decided handicap to vision because of the rapid sensory adaption shown by the retina. In addition to adaption, the retina would suffer still further from accurate fixation by the development of troublesome after-images. As a final objection to any theory which assumes that different portions of a zone can transmit different degrees of intensity over the single connecting fiber, it may be said that all recent work on sensory receptors has shown that they obey the all-or-none law similar to the nerve impulse in a motor fiber. There is every reason therefore to believe that fractional stimulation of a cone or even fractional conduction in a nerve fiber, is impossible.

Now it is a well known fact that when one is dealing with a large number of measurements, the accuracy obtained by averaging a large number of these, is

Thus far the problems of fixation have been discussed as they affect one eye. When some form of stereoscope is used (such as the stereocampimeter or screen of Goldman), not only do the movements of fixation of each eye need consideration but also the fact that the eyes will not maintain an absolutely constant angle between the respective visual lines (optical or actual). This is commonly noted when the routine tests for muscle balance are made (Maddox rod, red glass and screen and parallax tests) for near as well as for far vision. The swaying of the visual lines is so marked that the patient will remark about positional variation of the images without prompting. If an actual central scotoma exists, there is a deviation of the affected eye which brings the next most efficient area of seeing retina into line for fixation. This occurs most markedly when the unaffected eye is prevented from fixing. The stereoscope permits the unaffected eye to fix and yet encourages the affected eye to "aim" as if it were fixing normally. There are two exceptions to this: First, if the defect has existed for a number of years or was acquired in infancy, the affected eye cannot take up its normal position, in spite of the demand of the unaffected eye (as divergence, and rarely convergence, of blind or nearly blind eyes). Second, if there is a marked departure from normal muscle balance, prismatic correction must be made to assist the proper alinement. (In my experience this has seldom been satisfactorily accomplished, as the swaying movements are greatly exaggerated by the very mechanism which accounts for the great muscular imbalance and also by the loss of macular vision.)

With these points in mind, it thus becomes obvious that if one eye is occluded during a stereocampimeter examination, one variable and less controllable factor will be eliminated as a source of error. It is

actually greater, even though the physical measurement is done roughly, than if a few measurements were done with extreme care. In the same manner the shift in position of a linear image on the retina by stimulating a relatively large number of receptor units, each having its own local sign, may result in more accurate localization in consciousness of the line than can be accounted for by its shift in position from one cone to another. The length of the line according to this theory will materially determine the accuracy with which a shift in position can be visualized, since the longer it is, the more cone groups will be affected. Experiment shows this to be so. Within certain limits the alining power of the eye is considerably enhanced by increasing the length of the line."

As already noted, outlining the scotoma, as the examination progresses, is akin to calling forth the alining power of the eye. There is evidence of an effort of the eye to use the borders of the plotted defect as a secondary means of fixation. In fact, as these studies have progressed—using the minute object as a test—the conviction has been impressed on me with increasing force that the alining power of the eye is more responsible for the successful outlining of the defect than has been previously suspected.

only occasionally that "binocular fixation" (perhaps "conjugate fixation" would be a better term, as it indicates a yoking of one eye with the other during the act of fixation) seems to give better results in such cases if there is a very small central scotoma in one eye.

In the study of a small series of cases³¹ in which central retinitis of each eye produced a central scotoma in each field, the relative position of the fixation lines was the same as in normal eyes, apparently because these eyes were turned so that the acuity isopters of equal value were used for fixation instead of the macula. This is more likely to be so if the central scotomas are equal in size and degree (as they were in these cases). (As a rule one does not expect in this type of case to see deviation of the eyes long after the onset of the condition, but one does expect to if a central scotoma affects one eye only.)

31. There were only six cases (a rather insufficient group) for this study on account of the rarity of satisfactory conditions: (1) an atrophic stage of bilateral central chorioretinitis (2) which had developed in an adult after the full development of acuity, fixation and fusion, (3) without a previous demonstrable muscular anomaly or (4) nystagmus, (5) equal-sized atrophic lesions in the two eyes (measured with the graticule of a Keeler scope); equal visual acuity in the two eyes, and (6) satisfactory cooperation for mapping. In case 1 vision without correction in the right and in the left eye, respectively, was 6/15 +3 and 6/18; in case 2, 6/15 +1 and 6/18; in case 3, 6/15 +2 and 6/18; in case 4, 6/18 and 6/18; in case 5, 3/21 +2 and 3/21, and in case 6, 3/21 +2 and 3/21.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

UNIVERSITY NEWS

Dr. Don Marshall has been appointed full time assistant professor of ophthalmology at the University of Michigan Medical School.

SOCIETY NEWS

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat.—The following officers were elected for the coming year at the fourteenth annual meeting of the Section on Eye, Ear, Nose and Throat of the Connecticut State Medical Society, held at Hartford on May 21: Dr. Walter L. Hogan, Hartford, chairman; Dr. Charles T. Flynn, New Haven, vice chairman; Dr. Shirley H. Baron, New London, secretary-treasurer. Dr. Conrad Berens, of New York, addressed the section on "Clinical Considerations of Ocular Fatigue," and Dr. E. Ross Faulkner, of New York, spoke on "Review of Operative Technic in Nose and Throat Surgery."

North Dakota Academy of Ophthalmology and Oto-Laryngology.—At a recent meeting of the North Dakota Academy of Ophthalmology and Oto-Laryngology, held at Jamestown, the following officers were elected: Dr. J. P. Miller, Grand Forks, president; Dr. H. Rosenberger, Bismarck, vice president; Dr. F. L. Wicks, Valley City, secretary-treasurer. Dr. Erling W. Hansen, Minneapolis, visiting clinician, spoke at the meeting on "Allergy in Ophthalmology." Other speakers were: Dr. G. A. Larson, Fargo; Dr. G. M. Constans, Bismarck, and Dr. N. A. Youngs, Grand Forks.

GENERAL NEWS

Fifteenth International Ophthalmological Congress.—The Fifteenth International Ophthalmological Congress will be held in Cairo, Egypt, from Dec. 8 to 14, 1937. The two official subjects of the congress will be: "Arterial Hypertension of the Retina" and "Endocrinology and the Eye." Papers to be read in the scientific meeting should be sent directly to Dr. Marx, Oostzeedijk 316, Rotterdam. A subscription fee of 50 Swiss francs and requests for further information should be sent to the Secretary General, XV. Concilium Ophthalmologicum, P.O.B. No. 2001, Cairo, Egypt.

Summer Graduate Course in Ophthalmology, University of Rochester.—The department of ophthalmology of the University of Rochester School of Medicine and Dentistry will conduct the seventh annual summer graduate course of lectures and demonstrations in ophthalmology from July 27 to 31, 1936. The course is designed to offer a series of lectures and demonstrations of interest to all practi-

tioners in ophthalmology. Recent advances and observations in ophthalmology, as related to general medicine and surgery, will be stressed.

Ophthalmologic surgery, ophthalmoscopy and medical ophthalmology, perimetry and ophthalmic neurology, slit lamp microscopy, ophthalmic myology, pathology, retinoscopy and refraction will be covered by this course. The tuition fee for the course is \$40. Further information may be secured from Dr. John F. Gipner, Strong Memorial Hospital, Rochester, N. Y.

Course in Ophthalmology, New York Post-Graduate Medical School.—A course in "Motor Anomalies of the Eye, for Ophthalmologists" will be given by Dr. James W. White at the New York Post-Graduate Medical School, Columbia University, from July 13 to 19, 1936. Information regarding application and the registration fee may be secured from the director, 303 East Twentieth Street, New York.

Lucien Howe Prize.—Dr. Arthur J. Bedell, of Albany, N. Y., was awarded the Lucien Howe Prize by the Medical Society of the State of New York at its meeting in New York on April 28, 1936.

Obituaries

HENRY H. TYSON, M.D.
1865-1936

Dr. Henry H. Tyson, son of Henry H. and Leticia Tyson, died suddenly of heart disease at his home on April 18, 1936, at the age of 71.

Dr. Tyson was educated at the College of the City of New York and was graduated from the New York University Medical College in 1887. Immediately after graduation, he became associated with the ophthalmic department of the New York Ophthalmic and Aural Institute, where he worked under Dr. Herman Knapp. He continued his association with this same institution after it became the Herman Knapp Memorial Eye Hospital in 1913. In 1895 he was appointed surgeon, and he continued in that capacity until his death. He was also a clinical instructor in diseases of the eye at the College of Physicians and Surgeons from 1903 to 1915.

In addition to designing a tattooing needle which was used in the application of color to the cornea, Dr. Tyson was the discoverer of the Tyson-Clarke ocular syndrome in dementia praecox. He was one of the first to discover the relationship between tuberculosis and keratitis disciformis.

With Dr. M. J. Schoenberg, he presented before the American Medical Association in 1914 an exhaustive study on experimental researches on the inhalation of methyl alcohol. While Tyson did not contribute extensively to the ophthalmic literature, his papers were always well considered and practical, and his discussions were interesting and sound.

During the World War Dr. Tyson was special ophthalmic examiner for the third district of the New York Draft Board. He was ophthalmologist to the Sea-Breeze Hospital, in Brooklyn, and to Letchworth Village, Thiells, N. Y.

He was a fellow of the New York Academy of Medicine, and a member of the county and state medical societies, the American College of Surgeons and the American Ophthalmological Society. He joined the American Ophthalmological Society in 1906 and was one of its oldest members. He was also a member of the Medical Veterans of the World War, the Oxford Ophthalmological Congress and the New York Athletic Club.

He was a devoted member of the New York Ophthalmological Society for thirty years and rarely missed a meeting. He was president

of that society for one year and chairman of the Section of Ophthalmology of the New York Academy of Medicine for two terms.

Dr. Tyson had a long and successful career in the practice of ophthalmology and was greatly interested in the subject at all times. His sudden death came as a distinct shock to a host of medical friends and admirers. Although of a retiring disposition, he had by years of earnest and kindly living endeared himself to many friends both in and out of the medical profession, so that his passing is a great loss to the profession, to the community and to the hospital with which he had been connected for so many years.

Surviving him are two children, Mrs. Eleanor Tyson Bridgman and Henry B. Tyson, and several grandchildren.

GEORGE HUSTON BELL.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE STRUCTURE OF THE SCLERAL CANALS AND THEIR RELATIONS TO THE VENAE VORTICOSAE AT VARIOUS AGES. E. MARCHESINI, *Ann. di ottal. e clin. ocul.* 63: 689 (Sept.) 1935.

Twelve eyes with posterior segments normal except for the changes due to age were examined with especial reference to possible changes in the sclera with age. Two were from young adults aged 20 and 28, two from persons aged 51 and 59, and eight from persons aged from 60 to 87. Certain changes in the eyes were found, chiefly in persons past 60, and more advanced changes in those of greater age. These consisted in a greater compactness of the scleral structure, the fibers becoming thinner, more parallel and more closely opposed to each other, and a reduction in number and size of the fixed scleral cells. The sclera about the vortex veins became more dense, and in three eyes of persons of 70, 75 and 87 a special condensation of the sclera was seen about the veins, forming a sort of sclerotic cuff. The connective tissue, which in young eyes was arranged loosely about the veins so as to leave available a certain perivascular space for expansion, was much thinner or entirely absent in the older eyes, with a few exceptions. In only one eye was definite arteriosclerotic thickening of the venous wall observed. The findings confirm those of Weichselbaum and support the possibility that such changes, by removing a mechanism which allows the veins to expand when the intra-ocular tension is increased, may play a part in glaucoma.

S. R. GIFFORD.

Bacteriology and Serology

A STUDY OF THE PNEUMOCOCCUS GROUP FROM THE INFLAMED CONJUNCTIVA AND LACRIMAL SAC. S. H. MCKEE, *Am. J. Ophth.* 18: 1021 (Nov.) 1935.

This article does not lend itself to abstracting. It is based on the possibility that local and general serum treatment of patients with pneumococcic infections of the various types may lead to the establishment of a new and thorough medication.

W. S. REESE.

THE COLON BACILLUS IN OCULAR PATHOLOGY. J. FRANÇOIS, *Arch. d'opht.* 52: 424 (June) 1935.

Two cases of infection with the colon bacillus are described: one, a case of metastatic panophthalmitis; the other, of congenital dacryocystitis. Cultures were obtained and experiments made on rabbits. The conditions which have been found due to this organism are listed and briefly discussed. The results of the experiments conform to those

reported by other workers. The conclusion reached from clinical observation, confirmed by laboratory study, is that under certain conditions the colon bacillus is able to infect the eye directly and by metastasis. Its presence in the eye is exceptional.

S. B. MARLOW.

BIOLOGIC AND EPIDEMIOLOGICAL STUDIES ON STREPTOCOCCI OF THE EYE. S. SPIRATOS, *Arch. d'ophth.* 52: 505 (July) 1935.

Spiratos describes a case of metastatic ophthalmitis following streptococcic pneumonia, which he encountered during a study of many types of streptococcic inflammation of the eye. He could find reports of only four instances of a similar condition in the literature. In his case *Streptococcus viridans* was found in the vitreous and in the sputum, while *Streptococcus haemolyticus* was found in the conjunctival secretion. Culturing the blood gave no growth, probably because of the late stage at which it was done. In seeking an explanation for the occurrence of these two organisms in this case the question of transformation arises. This is discussed at some length, Spiratos leaning to the opinion that it is a better explanation in this instance than the coincidence of two different organisms. The occurrence of *Str. haemolyticus*, according to the observations made, is greatest during December and January. He points out that this is of special significance in relation to the surgical work done during this period.

S. B. MARLOW.

Biochemistry

SODIUM CONTENT OF AQUEOUS, VITREOUS AND SERUM: COMPARATIVE STUDY ON OXEN. P. W. SALIT, *Am. J. Ophth.* 18: 706 (Aug.) 1935.

Salit found the sodium contents of aqueous and vitreous to be identical and that of serum approximately 9 per cent greater.

W. S. REESE.

THE BLOOD-AQUEOUS BARRIER AND VITAMIN C: I. THE PERMEABILITY OF THE BLOOD-AQUEOUS BARRIER AND THE ASCORBIC ACID CONCENTRATION IN THE ANTERIOR CHAMBER. H. GOLDMANN and W. BUSCHKE, *Arch. f. Augenh.* 109: 205, 1935.

Large amounts of ascorbic acid (the reduced form of vitamin C) have been found in the normal aqueous of man and animals. Its concentration in rabbit aqueous is about 20 mg. per hundred cubic centimeters. The vitamin C in the aqueous comes, for the most part, from the metabolism of the lens. The lens is known to reduce the oxidized form of vitamin C which enters the anterior chamber from the blood stream; but, in addition to this, the lens itself probably forms small amounts of the vitamin. As a result the concentration of the vitamin in the aqueous is about ten times that in the blood stream. It is known that vitamin C can diffuse freely from the blood into the aqueous, but the high concentration found in the aqueous suggests the possibility that this permeability is not reversible and that the aqueous-blood barrier

normally prevents vitamin C from moving back into the blood stream from the aqueous.

The present experiments were designed to test this hypothesis. Rabbits were used throughout the experiments. The concentration of vitamin C in the aqueous was determined by Harris' method. Special pipets were used to withdraw the aqueous from the anterior chamber so that it was never in contact with air except in the pipet itself. A preliminary series of experiments proved the concentrations of vitamin C equal in the two eyes in a series of normal rabbits. The permeability of the blood-aqueous barrier was increased by injecting sodium chloride subconjunctivally in concentrations of from 5 to 15 per cent. An increase in the concentration of protein in the aqueous and a more rapid appearance of fluorescein in the anterior chamber indicate to the authors that the injection of hypertonic solutions of sodium chloride effectively increases the permeability of the blood-aqueous barrier. They found a reduction in the vitamin C content of the aqueous in the majority of cases in which the hypertonic salt solutions were employed. This reduction in the concentration of vitamin C did not run parallel with the increase in the protein content of the aqueous but followed more nearly the increase in the fluorescein in the anterior chamber. They regard the latter as a better indicator of permeability than the increase in the concentration of protein. Injections of epinephrine into a normal eye did not raise the concentration of vitamin C in the aqueous but, on the contrary, sometimes lowered it. Intramuscular injections of theophylline also lowered the concentration of vitamin C in the aqueous.

Goldmann and Buschke conclude that the blood-aqueous barrier normally prevents the reabsorption of vitamin C from the aqueous into the blood but that when the permeability of this barrier is increased vitamin C is rapidly reabsorbed into the blood stream. They discuss the possible relation which this may have in the production of cataract, more particularly cataracta complicata, in which they believe the inflammatory process (uveitis) may so increase the permeability of the blood-aqueous barrier that the vitamin C necessary for the metabolism of the lens disappears from the aqueous and the lens becomes opaque.

F. H. ADLER.

Conjunctiva

THE RETICULUM IN THE CHRONICALLY HYPERPLASTIC CONJUNCTIVA.
H. D. LAMB, *Am. J. Ophth.* 18:724 (Aug.) 1935.

Lamb found fairly uniform changes as regards reticulum in the hyperplastic conjunctiva in cases of trachoma with the exception of those involving the cornea. Comparing his observations with those of Beigelman on folliculosis, he concludes that it is difficult to see how the demonstration of reticulum in the hyperplastic conjunctiva offers any assistance in the early diagnosis of trachoma. Similar changes were also seen in a case of chronic lymphatic leukemia.

W. S. REESE.

THE CONJUNCTIVITIS OF SCARLET FEVER. H. OTTO, *München. med. Wchnschr.* 82:1987 (Dec. 13) 1935.

The majority of the infectious diseases begin with conjunctivitis. The teaching in the past has been that tearing, photophobia and redness

of the conjunctiva are not a part of scarlet fever. Otto's experience has been otherwise. In the Halle-Wittenberg clinic during the years from 1927 to 1935, more or less definite conjunctivitis was noted in 52, or 5.8 per cent, of the 891 cases of scarlet fever. Otto divides the types as follows:

I. Bilateral conjunctivitis

1. Conjunctivitis of the bulb with fine injection of the superficial blood vessels without photophobia, tearing or heaviness
2. Conjunctivitis of the bulb with violent diffuse injection of blood vessels
3. Diffuse fiery-red inflammation of the bulb with and without photophobia, tearing and heaviness
4. Forms 1 to 3 with more or less marked edema
5. Tarsal and bulbar conjunctivitis of grades 1, 2 and 3 with and without photophobia and tearing but with blepharospasm
6. Blepharoconjunctivitis
7. Pure blepharitis with and without marked edema
8. Angular conjunctivitis—medial
9. Angular conjunctivitis—lateral
10. Angular conjunctivitis with more irregular situation
11. Mild conjunctivitis which later becomes marked and also returns periodically

II. Unilateral conjunctivitis

1. The forms classified under I may affect only one eye
2. Unilateral conjunctivitis which later involves the other side

III. Complications

1. Perforation of the globe with prolapse of the iris, fiery-red injection of the tarsus and bulb with blepharitis and edema of the lid
2. Secondary corneal inflammations
3. Phlyctenules of the cornea in connection with those of the conjunctiva

The early conjunctivitis presents itself from the first to the sixth day of the disease, usually together with lesions of the skin and occasionally before these. It remains for from two to sixteen days. The late conjunctivitis occurs between the fifteenth and the fiftieth day and lasts from eight to twenty-five days. In only four cases of scarlet fever seen by Otto was rheumatism present with conjunctivitis. Because of the finding of an increase in the number of eosinophils in the blood there is some reason to believe that the conjunctivitis of scarlet fever may be an allergic reaction. It also may be due to the toxins of the organism causing the scarlet fever. The hemolytic streptococcus is found in the conjunctival sac no more frequently than other organisms, and therefore the possibility of a direct infection or of one due to transference through the tear passages is not to be considered.

L. L. MAYER.

Cornea and Sclera

"PALM LEAF" OPACITIES OF THE CORNEA: RESIDUALS OF INTERSTITIAL KERATITIS (ANNULAR TYPE OF VOSSIUS). P. BONNET, *Arch. d'ophth.* 52: 625 (Sept.) 1935.

The disposition of these opacities in the deeper layers of the cornea produces a figure which is best compared to a palm leaf. They are encountered rather frequently and have been known for some time, illustrations of them having appeared in the first edition of A. Vogt's "Atlas of the Slitlamp-Microscopy of the Living Eye" and in a paper by Gallemaerts. However, Bonnet suggests that these opacities have not been sufficiently individualized as residua of interstitial keratitis. He describes the opacities in some detail and with extensive illustrations. They are seen only on examination with a slit lamp. They are present on both sides and are more frequently seen in elderly women, although he has observed their development in a 13 year old girl with active interstitial keratitis. Although it is possible that they may occur in other forms of deep keratitis, they are especially significant signs of the occurrence of interstitial keratitis many years before. They constitute, so far as present knowledge permits, an indelible stigma of hereditary syphilis.

S. B. MARLOW.

THE SYNDROME OF BLUE SCLERAS. C. CORDERO, *Arch. di ottal.* 42: 521 (Dec.) 1935.

This concludes an article previously reviewed in part, which constitutes a complete monograph on the subject of blue scleras. Cordero here discusses certain phenomena associated with the condition aside from the well known fragilitas ossium. Deafness, the third symptom in the syndrome described by van der Hoeve, Cordero noted in two of his patients, and in the others, abnormalities of the vestibular mechanism, usually hyperirritability. Only one showed the blue discoloration of the tympanic membrane which has been described in a few cases recorded in the literature. Syndesmolyis, or frequent dislocation of various joints, was present in all his cases. Neuromuscular changes were present in all, consisting in hyperirritability as shown by electric tests. None of the patients showed psychic abnormalities. One showed marked diminution in the elastic tissue of the skin. Two patients showed anomalies of the teeth, one having lost all the teeth at an early age, and all gave a history of late eruption of the teeth. Fragility of the nails and loss of hair were observed in most patients. The blood calcium content was normal, as were other findings of the blood chemistry except that cholesterol was increased in all but one of the patients, reaching 400 mg. in two. The dominant hereditary character of the syndrome was evident in Cordero's family, seventeen of thirty-eight members being affected. After a review of various theories, he gives his reasons for believing that hypoplasia of both mesenchymal and ectodermal tissues must be present to account for all the changes observed in the syndrome.

S. R. GIFFORD.

AN UNUSUAL FAMILIAL SUPERFICIAL CHANGE IN THE CORNEA:
REPORT OF A CASE. J. K. PAMEIJER, *Klin. Monatsbl. f. Augenh.*
95: 516 (Oct.) 1935.

A boy aged 8 had a high degree of hypermetropia. The vision in his right eye, 2/60 of the normal, could not be improved with glasses, whereas the vision of 3/4 in the left eye was brought up to 5/4 by a convex lens of 8 diopters. The eyes were free from irritation, and the sensitiveness of the corneas was normal. The corneal microscope revealed that the superficial layers of the corneas were strewn with minute and fine gray spots and dots. Some of them were round; others had the shape of a pointed angle. The peripheral region of the cornea, near the limbus, showed only a few isolated dots. A narrow strip in the nasal and lower quadrants of each cornea was entirely clear in the boy and in four other members of the family. The form and size of these strips varied in the five patients. The dots appeared to be minute vacuoles or blisters with a double outline and either a clear center or a clear periphery. The substantia propria and the endothelium were normal.

The mother presented similar but larger vacuoles, some of which stained green with fluorescein; she complained of epiphora and photophobia for many years. The mother, her sister and two of the latter's four children had the peculiar clear strips. No connection existed between the clearly visible corneal nerves and the opacities. Two brothers of the mother were allegedly afflicted with the same malformation; they could not be reached for an examination. Green staining was obtained only in the two women, not in the children. Epithelial dystrophy (Fuch's) and nodular and lattice-shaped degeneration did not exist, nor were symptoms of avitaminosis present.

K. L. STOLL.

REMOVAL OF FOREIGN BODIES FROM POSTERIOR LAYERS OF THE CORNEA.
G. G. BURSUK, *Sovet. vestnik oftal.* 7: 154, 1935.

Bursuk constructed a knife for removal of amagnetic foreign bodies from the posterior layers of the cornea when routine measures are of no avail. The knife, when entered into the anterior chamber, adheres firmly to the posterior surface of the cornea and thus supports the foreign body and prevents it from slipping into the anterior chamber while it is being obtained from the external surface. It also prevents any possible injury of the iris or lens.

A description of the knife and of the technic of its use is given. It was tried out successfully in several cases in Bursuk's clinic. The slit lamp is essential for localization of the foreign body.

O. SITCHEVSKA.

Experimental Pathology

EXPERIMENTAL PTOSIS IN PRIMATES. W. MAHONEY and D. SHEEHAN,
Arch. Neurol. & Psychiat. 35: 99 (Jan.) 1936.

This study was prompted by an incidental observation in monkeys of a rapid recovery from the ptosis that follows experimental section of

the third nerve. The authors show that Müller's muscle, through sympathetic innervation, overcame the ptosis produced by intracranial section of the third nerve in from two to three weeks in monkeys and in a much shorter time in cats and dogs. After section of the nerve, excitement caused excessive widening of the palpebral fissure, presumably because of lack of inhibition from the third nerve.

After removal of sympathetic influence in addition to section of the third nerve, complete closure of the lid persisted until regeneration of some fibers of the third nerve had occurred. This regeneration was demonstrated by anatomic studies and was found to be nearly complete in three months.

After section of both the third and the sympathetic nerve it was observed that extreme fright or injections of epinephrine caused widening of the palpebral fissure, greater on the side of the operation than on the intact side. This paradoxical response is compared by the authors to the paradoxical dilatation of the pupil after section of the sympathetic nerves reported by Byrne and others. It is presumably the result of the action of epinephrine on a neuromuscular mechanism sensitized through being deprived of its sympathetic nerve supply.

Section of the seventh nerve and observation of paradoxical responses after degeneration of the sympathetic nerves over a longer period than is reported here would be of additional interest. However, the presentation of the work that has been done is pleasingly concise and scientifically instructive.

R. IRVINE.

General

OPHTHALMOLOGY IN THE WORLD WAR. A. JESS, München. med. Wchnschr. 82: 1941 (Dec. 6) 1935.

During the war of 1870-1871, only 0.86 per cent of the injuries were injuries of the eyes; in the Chinese-Japanese war of 1894, only 1.18 per cent; in the Russo-Japanese war of 1904, only 2.22 per cent, while in the World War the figure reached 10 per cent. It was due to this fact that field stations for the treatment of ocular injuries were established, and many new and unusual procedures in the management of injured eyes were of necessity carried out by ophthalmologists. Fear of sympathetic ophthalmia caused them to enucleate many eyes. The necessity of having a strong magnet in the stations for removal of metallic foreign bodies from the injured globes was emphasized. Hemorrhage into the globe, luxation of the lens, rupture of the iris and choroid, detachment of the retina and other obscure injuries due to force were often entirely overlooked because of the general condition of the soldiers. Injuries to the brain in some cases caused blindness by interference with the centers of vision. The cornea and conjunctiva were often affected by gaseous fumes. Burns from explosive shells occurred frequently. Methyl alcohol poisoning was seen with its primary attack on the papulomacular bundle. Many chemicals were used by the soldiers in attempts to cause injury to their own eyes and thus escape service. Alum, soap, tobacco, paprika, chalk and other substances were tried. Night blindness due to avitaminosis was of frequent occurrence. Several cases are cited. Trachoma in its various phases was present and was

often adduced to gain exemption from service. Refraction and correction of the numerous near-sighted persons in the German army frequently called for the ingenuity of the oculist. Color blindness prevented proper recognition of color signals. Additional diseases encountered were all the more or less harmless ophthalmic conditions seen in every day life. In Germany 3,200 were blinded during the period of the war.

L. L. MAYER.

General Diseases

THE FUNDUS OCULI IN DIABETES MELLITUS. M. L. FOLK and S. SOSKIN, *Am. J. Ophth.* 18: 432 (May) 1935.

Folk and Soskin give the following summary and conclusions:

"1. A study of the fundi of 150 diabetic patients and of a control group of 150 subjects without diabetes is reported.

"2. The correlation of certain retinal conditions, with the age, duration, and severity of diabetes and diabetic complications is brought out.

"3. The incidence of retinitis in those affected with diabetes is greater than is commonly believed.

"4. Diabetic retinitis is a definite entity and occurs in diabetes in the apparent absence of its complications.

"5. The incidence of sclerosis of the retinal vessels is but little higher in persons with diabetes than in those without in the same age groups, and when present is usually less severe.

"6. The prognosis of diabetic retinitis is comparatively good, provided the patient is properly managed and cooperates well.

"7. Effective diabetic therapy should lessen the incidence of retinitis.

"8. Frequent fundus examinations are of particular value to the diabetic patient."

W. S. REESE.

POSTGRIPPAL DISEASES OF THE EYE. S. M. KHORDAS, *Sovet. vestnik oftal.* 7: 305, 1935.

Khordas selected a group of thirty-one patients whose eyes were affected during a severe epidemic of influenza in the years 1930 and 1931. Most of the patients were from 20 to 30 years old; the male sex prevailed. The cornea was most frequently affected, usually in the period from the seventh to the eleventh day after the grip. The clinical picture characteristic of herpetic keratitis was usually observed: photophobia, diminution of corneal sensitivity, ciliary injection and the presence of a number of small vesicles along the limbus. There were also recorded two cases of iritis, one of iridocyclitis, two of tenonitis, two of scleritis and three of unilateral paralysis of the abducens nerve.

Khordas believes that all postgrippal ocular complications are caused by the virus of herpes, that the virus of grip, by reducing the resistance of the body, activates the virus of herpes, and that thus complications of the eye set in.

Daily intravenous injections of a 40 per cent solution of methenamine (urotropin), because of the good therapeutic results from the use of

this drug in encephalitis, were tried in the treatment of these complications of the eye. The initial dose was 2 cc., and the amount was increased daily until a dose of 7 or 8 cc. was reached; the total number of injections varied from fifteen to twenty-five. The corneal disorders responded well to the treatment, as the sensitivity was restored and healing of the infiltrates took place after the first few injections.

O. SITCHEVSKA.

Glaucoma

EPINEPHRINE BITARTRATE: USES OTHER THAN IN THE TREATMENT OF GLAUCOMA. F. C. CORDES and D. O. HARRINGTON, *Am. J. Ophth.* 18:451 (May) 1935.

Cordes and Harrington draw the following conclusions: "Epinephrine bitartrate is a relatively little-used drug which deserves a prominent place in the ophthalmologic pharmacopeia.

"In addition to its action of lowering tension in chronic simple glaucoma, it has been found useful as a mydriatic for:

"1. Examination of the fundi in eyes with high normal tension.

"2. Examination of the fundi in glaucomatous eyes which are under the influence of a miotic.

"3. Powerfully dilating the pupil with the separation of posterior synechia in cases of old or neglected iritis.

"4. Dilating the pupil during the operation for congenital, soft, membranous or traumatic cataract.

"The marked vasoconstrictive action of this drug, which exceeds that of adrenalin, makes it an ideal hemostatic in the removal of chalazia, in cyclodialysis, and in the operation for retinal detachment, in which a dry field is essential to success."

W. S. REESE.

A CRITIQUE OF GLAUCOMA OPERATIONS. H. S. GRADLE, *Am. J. Ophth.* 18:730 (Aug.) 1935.

This paper does not lend itself to abstracting. Gradle considers operations for glaucoma in three divisions: (1) those to restore normal paths of drainage, (2) those to open new paths of drainage and (3) those to form paths of extra-ocular drainage. He concludes with a description of a new procedure to be used in the treatment of absolute glaucoma, namely, diathermic coagulation of the vitreous. He has used this in six cases, and in none was there recurrence of pain although the reduction of tension was only temporary.

W. S. REESE.

LATE INFECTION FOLLOWING A FISTULIZATION OPERATION FOR GLAUCOMA. D. CATTANEO, *Ann. di ottal. e clin. ocul.* 63:481 (July) 1935.

After a fairly complete review of the literature, Cattaneo describes a case in which infection developed eighteen years after a trephining for secondary glaucoma. The anterior chamber became filled with pus, and a ring abscess of the cornea developed rapidly. Pneumococci were found in smears and cultures of aspirated aqueous. Sections of the enucleated

eye showed the same organisms in the vitreous, and they were also present in the conjunctival secretion. Sections showed the route of infection from the conjunctiva over the trephine opening to the corneal stroma and intra-ocular contents.

S. R. GIFFORD.

THE RISE IN INTRA-OCULAR PRESSURE IN ACUTE ANGIONEUROSIS OF THE CILIARY BODY (GLAUCOMA ALLERGICUM) AND ITS RELATION TO CYCLITIC AND HETEROCHROMIC GLAUCOMA. ERNST KRAUPA, Arch. f. Augenh. 109: 416, 1935.

By serous cyclitis one refers to a condition characterized by many opacities of the vitreous and deposits on the posterior surface of the cornea without the formation of posterior synechiae or visible nodules in the iris. It is frequently associated with a rise in intra-ocular pressure (secondary glaucoma). Frequently after the pressure has been reduced one may find central or peripheral areas of choroiditis. The disease is nearly always of tuberculous origin. With successful management of the secondary glaucoma the disease can nearly always be cured by foreign protein therapy and by the use of the roentgen rays. After the condition has been present for some time the iris as a rule becomes discolored so that it is difficult to differentiate the condition from the so-called Fuchs type of heterochromia.

In the latter condition miotics are also not indicated, as the glaucoma disappears if the anterior chamber is opened. Although Kraupa admits that the cause of Fuchs' heterochromia is not known, he considers in a rather vague way that it is due to some constitutional disturbance in the metabolism of the ciliary body. He likens this disease to constitutional albuminuria. A general discussion of heterochromia is given, with particular emphasis on Passow's views. The latter considers Horner's syndrome as a motor disturbance, heterochromia as a trophic disturbance and Fuchs' heterochromia as a vasomotor disturbance in the sympathetic nervous system, and likens these to masked forms of syringomyelia.

Four cases of glaucoma and cyclitis are reported, and a rather confused general discussion follows as to the possible relation between these two pictures. Kraupa looks on these cases as representative of a group in which, through some vasoneurotic disturbance of the ciliary body, protein is poured into the aqueous, with resulting increase in pressure. He prefers to use the term "glaucoma allergicum" for this condition.

F. H. ADLER.

Hygiene, Sociology, Education and History

ILLUMINATION INTENSITIES FOR READING. M. A. TINKER, Ann. J. Ophth. 18: 1036 (Nov.) 1935.

Tinker reviews the work done on intensities of illumination for reading. He concludes that in the light of experimental data the following specifications for intensities of light should fulfil the requirements of hygienic vision for the reading of legible print by the normal eye: 3 to 5 foot-candles with direct lighting and poor diffusion; 5 to 10 foot-

candles with the combinations of direct and semi-indirect illumination frequently found in homes and offices; 10 to 15 foot-candles with the better degrees of diffusion found in a few homes and offices. For defective eyes and the discrimination of fine details the intensities should be much higher.

W. S. REESE.

Lacrimal Apparatus

STRICTUROTOMY AND PROBING IN THE TREATMENT OF LACRIMAL DISEASE. R. SÁENZ-ALONSO, *Arch. de oftal. hispano-am.* 35:449 (Sept.) 1935.

VALUE OF CONSERVATIVE TREATMENT IN LACRIMAL DISEASES. MÁRQUEZ, *Arch. de oftal. hispano-am.* 35:455 (Sept.) 1935.

These two papers, published together, plead against the systematic discard of this method of treatment in appropriate cases, Sáenz-Alonso proposing several modifications in the procedure recommended by Poulard and Morax.

C. E. FINLAY.

TREATMENT OF EPIPHORA WITH ALCOHOL INJECTED INTO THE LACRIMAL GLAND. P. E. TIKHOMIROV, *Sovet. vestnik oftal.* 7:452, 1935.

The treatment was applied in stenosis of the duct or in hyperfunction of the lacrimal gland in which routine measures were of no avail. From 0.3 to 0.7 cc. of an 80 per cent solution of alcohol was injected into the palpebral part of the lacrimal gland. The average number of the injections varied from one to three. Edema of the lids and conjunctiva was observed for from three to five days. Ptosis usually persisted for from ten to fifteen days. Some patients complained of anesthesia of the temple and lids for a few days. Seventy-two patients (80 eyes) were treated with injections of alcohol for a period of two years; the patients were observed for from one to twelve months. Favorable results were obtained in 51 per cent. In thirteen patients there was a complete recurrence of the epiphora.

Alcohol was injected into the lacrimal glands of three dogs. The glands of the experimental dogs and one human gland were extirpated thirty-five days after the injections and examined microscopically. None of the four glands showed atrophy of the tissue. Tikhomirov therefore thinks that the alcohol affects the secretory nerves and causes a decrease in the function of the lacrimal gland. This explains the positive results observed as soon as three days after the injections and the recurrence of the epiphora, which could not recur in an atrophied gland. He states the following conclusions: 1. The injection of alcohol into the lacrimal gland is harmless. 2. In the treatment of recurrent epiphora repeated injections can be given. 3. Positive results were observed in 51 per cent of the cases. 4. Further observations are necessary to judge whether these results are permanent.

O. SITCHEVSKA.

Lens

TREMULOUS LENS. A. W. D'OMBRAIN, Brit. J. Ophth. 20: 22 (Jan.) 1936.

Iridocyclitis developed in a man 50 years of age with an almost mature cataract. With the slit lamp the lens was found to be tremulous. The iris was not tremulous, so there was no dislocation of the lens. On the slightest movement of the eye the lens trembled as if it were suspended on an elastic string. The phenomenon is attributed to fluidity of the vitreous.

The author could find no record of a similar case in the literature.

W. ZENTMAYER.

COMMENTS ON ONE HUNDRED AND FIFTY ATTEMPTS AT SYSTEMATIC INTRACAPSULAR EXTRACTION OF CATARACT. H. LAGRANGE, Ann. d'ocul. 172: 1003 (Dec.) 1935.

Lagrange, in 1930, in *Le bulletin médical*, published an article relative to an inquiry as to the best procedure for the extraction of cataracts. He now reviews the present technic, and, as in his published article on sclerotomy and on the operative treatment of strabismus, his comments have as their objective a research on simple technic. He states that it was the Arruga forceps that enabled him to proceed with this investigation.

He pictures (1) the circumstances that lead to faults in technic and (2) the operative results, and here dwells particularly on the post-operative complications. The faults to which he has alluded consist in an inability to grasp the capsule, the total extraction of a broken capsular sac not accompanied by its contents and the appearance of the vitreous body. The operative results are tabulated—first those in a series of sixty-one cases of intracapsular extraction combined with iridectomy in which the keratotomy was done with the Graefe knife. In a second series of forty-two cases, the keratotomy was done by puncture. In the third series of forty-seven cases, there was no iridectomy, and the keratotomy was performed by puncture. The visual results and other details are given for each of these series. The article has nine excellent illustrations.

S. H. McKEE.

PERFECTED CORNEAL SUTURE FOR EXTRACTION OF CATARACT. S. BALDINO, Rassegna ital. d'ottal. 4: 538 (July-Aug.) 1935.

Kalt's and Liégard's methods of suture, carried out before corneal section, are made unsatisfactory by a resulting depression of the edges of the wound.

Williams' and Czermak's methods of suture, employed after corneal section, fail to get perfect apposition of the edges of the wound.

Baldino passes his needle through the corneosclera before corneal section. The needle is not drawn through; it remains in situ. The Graefe section is then made as usual, but only up to the needle. The latter and its thread are then pulled through. With a sharp hook the corneal section at the upper pole is completed, and the thread between

the edges of the wound is seized by a blunt hook and pulled out in a loop large enough to allow extraction of the cataract. It is then tied, thus securing perfect anatomic apposition.

V. R. SYRACUSE.

Lids

OPERATION FOR ENTROPION AFTER VOGT'S METHOD. H. SCHLÄPFER, *Klin. Monatsbl. f. Augenh.* 94: 610 (May) 1935.

Vogt practiced for many years, but never before published a report on, a simple operation for spastic entropion. It is based on the principle of replacing the margin of the lower tarsus, turned faultily toward the eyeball, in its normal position. This is done by pushing the margin of the upper tarsus off the eyeball by pulling the temporal extremity of the lower lid in front of the upper lid. Thus the margin of the upper lid is placed between the margin of the lower lid and the globe at the temporal canthus. The older methods were devised with the aim of bringing the margin of the lower lid toward the globe so that the entire inner surface of the tarsus was in close contact with the globe after the operation. A liberal canthotomy is done down to the bone at the temporal canthus after the lids are pulled apart. Then a suture is laid through the external portion of the upper lid and out through the conjunctiva and the bulbar portion of the lower lid so that the suture appears in the bulbar portion of the canthotomy wound of the lower lid. After the suture is tied the wound surface of the upper lid must touch the conjunctival surface of the lower lid, the ventral margin of the wound of the upper lid must touch the dorsal margin of the wound of the lower lid, and the former canthus of the upper lid is placed behind the lower lid. This method is recommended for extensive entropion and those cases in which previous operations have failed.

K. L. STOLL.

Methods of Examination

NEWER DEVELOPMENTS IN PHOTOGRAPHY OF THE EYE. W. A. MANN, *Am. J. Ophth.* 18: 1039 (Nov.) 1935.

Mann discusses external photography and photography of the fundus. Under the former he takes up black and white representation, color and infra-red photography, and under the latter, moving pictures and color work. He favors the Finlay color method and enumerates its advantages. Infra-red photography of the fundus has not been satisfactory.

W. S. REESE.

SPECTRUM ANALYSIS AS A RELIABLE METHOD IN PROVING THE PRESENCE OF METAL IN AND AROUND THE EYEBALL: REPORT OF A CASE. ROLF SCHMIDT, *Klin. Monatsbl. f. Augenh.* 95: 440 (Oct.) 1935.

Only a few cases have been reported in which the presence of metal foreign bodies in the eyeball and in its neighborhood was proved by means of spectrum analysis. Schmidt adds the case of a man who suffered an injury to his left eye during the war in 1918. Amaurosis fol-

lowed, and chronic inflammation made the removal of the eye necessary in 1925. The patient complained of headache and of pain in the orbit for about two years, during which a 1 per cent solution of silver nitrate was used by another oculist. This treatment was discontinued when a slight brownish discoloration developed in a limited area of the conjunctiva. The oculist accused the patient of secretly using some coloring substance when the size of the brown discoloration increased. It had a diameter of 9 mm. when Schmidt admitted the patient to the University Eye Clinic in Freiburg. No pathologic signs could be observed in the conjunctival sac aside from slight congestion. The roentgenogram, however, revealed two foreign bodies, each the size of a pinhead; one was located near the base of the zygomatic process; the other, in the nasal portion of the roof of the orbit. A piece of the discolored conjunctiva was excised; it contained no melanosis or silver. Spectrum analysis of a small portion of conjunctival tissue proved strongly the presence of the silver lines 3281 and 3383 Å and less distinctly that of the copper lines 3247 and 3274 Å; furthermore, it revealed traces of calcium, probably remnants of dust, and traces of iron, which may have been the residue of blood. Argyrosis was therefore present, although the other tests had failed to discover it. The presence of the copper lines was explained by the common admixture of copper in silver nitrate pencils. The patient was convicted of having used a silver nitrate pencil to obtain a higher compensation as a war veteran.

K. L. STOLL.

Neurology

ENCEPHALOTRIGEMINAL ANGIOMATOSIS. M. APPELMANS, *Arch. d'ophth.* 52: 835 (Dec.) 1935.

That there is a distribution of cutaneous angiomas along nerve supplies in some instances has been known for a long time, but the association of "trigeminal nevi" with intracranial lesions was first pointed out by Kalischer in 1897. These nevi are often accompanied by glaucoma. The detailed description of a personal observation is presented, with photographs. The origin of nevi is discussed from an embryologic point of view and in relation to the sympathetic innervation. A table of twenty-four cases demonstrates the frequency of homolateral glaucoma and other ocular complications. According to van der Hoeve, Lindau's, Bourneville's and Recklinghausen's diseases belong in the same group. The differentiating factor is the embryologic stage at which they start. In conclusion, Appelmans believes (1) that angiomas of the area of the skin supplied by the upper two branches of the trigeminal nerve are very often associated with glaucoma and intracranial angioma; (2) that ocular and intracranial lesions co-exist frequently with nevi of the face which present no clearcut disposition in relation to nerve roots; (3) that the term "trigeminal angioma" of the face is inadequate, and "neurocutaneous angiomatosis" is more exact; (4) that roentgenograms of the skull should be made in all cases of extensive angiomatosis of the face, and (5) that the glaucoma associated with angiomatosis is of neurovascular origin.

S. B. MARLOW.

A NEW PROOF OF PERIMETRIC POLYMORPHISM IN OPTICOCIASMAL ARACHNOIDITIS. J. MALBRÁN, Arch. de oftal. de Buenos Aires 10: 634 (Aug.) 1935.

This is the report of a case in which a limitation of the visual field of the right eye to the upper nasal quadrant (with good central vision) and an irregular defect in the horizontal meridian over the temporal portion of the macular region in the left eye led the author to diagnose erroneously a meningioma of the tuberculum sellae ossis sphenoidalis. An operation revealed an opticochiasmal arachnoiditis, more pronounced around the right optic nerve. Death occurred in coma forty-eight hours after the operation. No intracranial explanation was found at autopsy.

C. E. FINLAY.

Orbit, Eyeball and Accessory Sinuses

PHYSIOLOGICAL CONSIDERATIONS IN THE TREATMENT OF PULSATING EXOPHTHALMOS. G. M. DORRANCE and P. E. LOUDENSLAGER, Am. J. Ophth. 17: 1099 (Dec.) 1934.

Dorrance and Loudenslager have studied the original reports of cases recorded since 1908 and comment on these. They discuss blood currents and blood vessel changes associated with arteriovenous fistula. They remark the reversal of current in the external carotid after ligation of the common carotid as not being generally known. They decry preoperative compression of the carotid vessels and simultaneous ligation of the internal jugular vein. In the presence of infection of the mouth, nose or throat the cervical wound should be drained. Cerebral accidents following ligations are largely due to sudden extreme reductions in blood pressures, resulting in stagnation of the blood in cerebral areas. They reach the following conclusions: "An arterio-venous communication between the internal carotid artery and the cavernous sinus should be expected to exhibit the same phenomena of hemodynamics observed in arterio-venous communications elsewhere in the body.

"On the basis of such expectation, ligation of the proximal artery—in this situation, the internal carotid—would produce a reflux of blood from the artery distal to the fistula, keeping active the fistula and possibly causing cerebral complications.

"Ligation of the common carotid, on the other hand, should be recognized as a partial ligation only of the internal. Because of the reversal of flow in the external, the internal becomes a branch of the external with about fifty per cent reduction in volume flow.

"Fractional ligation of the internal carotid in this manner presents a more satisfactory record chiefly from the standpoint of fewer cerebral sequelae. We submit that this may be accounted for by the probability that the partial reduction in blood pressure in the internal carotid is insufficient to start a backflow from the distal artery. And, further, if this backflow does not occur, the vessel will shrink in size, reducing its lumen and thereby reducing the size of any fistula present."

W. S. REESE.

BILATERAL ORBITAL TUMOR WITH EXOPHTHALMOS CURED BY MERCURIAL TREATMENTS. M. KALT, Arch. d'opht. 62: 655 (Sept.) 1935.

An adult, 35 years old, previously well, acquired in the space of a month bilateral exophthalmos with complete immobility of the eyeballs and lagophthalmic keratitis due to a tumor involving both orbits. One series of mercurial treatments failed to check the progression of the exophthalmos on the right side. Bilateral biopsy showed a chronic inflammatory condition of the tissue, which was dense fibrous tissue with a perivascular infiltration of lymphocytes. On further intensive mercurial treatment the masses subsided. Inflammatory tumor of the orbit is not uncommon, but it is exceptional to see it bilateral and symmetrical. Kalt believes his case to be one of orbital syphiloma with osteoperiosteal localization and sclerogummatous invasion of the orbit, the point of origin being in the lacrimal glands. The Wassermann tests were repeatedly negative. The effect of antisyphilitic treatment was delayed. Biopsy was extremely important in the management of the case.

S. B. MARLOW.

PULSATING AND INTERMITTENT EXOPHTHALMOS AND ITS NONSURGICAL TREATMENT. M. DE-PETRI, Riv. oto-neuro-oftal. 12: 306 (March-April) 1935.

The diagnosis of exophthalmos due to orbital varices is not difficult in the typical case. If the condition is due to a cavernous aneurysm of the carotid artery, the diagnosis is more difficult, especially if there is no bruit. This may lead to improper therapy.

Therefore, instead of having first recourse to surgical treatment, De-Petri makes a deep intra-orbital and perivascular injection of 2 cc. of a 10 per cent solution of quinine and urethane in the direction of the superior ophthalmic vein. One must be sure that one does not have the needle in a vessel, that the technic indicated is closely followed and that one is familiar with the possible complications and with the results that should follow.

This method obviates the dangers and complications of surgical intervention; it is within reach of every specialist and is devoid of danger if properly carried out. It may be applied in many cases. In case of failure, surgical intervention is always in order.

V. R. SYRACUSE.

Physiology

NEW THEORY OF VISION OF RELIEF: THE PRIORITY OF PERIPHERAL POINTS OF THE RETINA. ALAERTS, Arch. d'opht. 52: 320 (May) 1935.

In a previous communication Alaerts attempted to demonstrate the importance of certain points in the retina in vision of relief. In the present report he attempts to show that the peripheral points are of greatest importance in this function and describes some simple experiments which support his theory. He describes a case in which a patient reported that he had noticed a spot in front of his eye, close to him. The

year before he had noticed a spot some distance away. Ophthalmoscopic examination revealed a fresh hemorrhage in the temporal half of the retina and a pigmented patch in the nasal half. This observation is used to support his theory that the nasal portion of the retina gives rise to the impression of distance and the temporal half to that of things close by.

The conclusions which he draws from the observations he has made are: 1. The essential factor in vision of relief is the peripheral part of the retina. The macula acts as a point of reference. 2. The nasal part of the retina gives rise to the impression of distance and the temporal to that of nearness. 3. There is always a comparison by superposition of two differently named portions of the retina. 4. The portion of the retina where the visual impression is most centrifugal gives rise to the sense of relief. This is the priority of peripheral points.

The phenomenon of pseudoscopic relief is also used in support of the theory. This paper should be read in the original.

S. B. MARLOW.

NONGLAUCOMATOUS COLORED RINGS OR CIRCLES. A. POLACK, Bull. Soc. d'opht. de Paris, July 1935, p. 396.

The patient is a distinguished astronomer and therefore should be able to describe the phenomenon very clearly. Druault has attributed the colored circles envisioned by persons without glaucoma to changes in the lens. The patient described the same distribution of colors and the same radial striations of the same short diameter as glaucomatous patients have noted, except that the visibility was not augmented by mydriasis. Also the disposition of the fishing hooks which are noted when a stenopaic slit is used were not eccentric from the pupil. These hoops which Druault calls "segments of a circle" and which are presented clearly when the subject looks at a point of light are at the periphery of the visual fields. In Polack's case these were noted in the center of the field at the luminous point. They were not seen when the stenopaic slit was deviated from the center line of the pupil. The patient, aged 75 years, had an increase of myopia from 4.5 to 6.5 diopters in one year's time. The visual acuity remained the same. The fundus was normal. In the vitreous were floaters and a striated membrane at the pupillary plane. No hypertension existed even after the use of atropine. The cornea, aqueous and lens were normal. Perhaps the changes in the vitreous account for the colored circles? The work of Rochon-Duvigneaud on the structure of the lens may aid in comprehending the mechanism.

L. L. MAYER.

THE DURATION OF AFTER-IMAGES. J. ESCHER-DESRIVIÈRES and R. JONNARD, Compt. rend. Soc. de biol. 120: 681, 1935.

The observer, after being in obscurity for a half hour, viewed through a binocular photographic shutter for one-tenth second a square of transilluminated opal glass and by a stop-watch subjectively measured the duration of his after-image. Under definite conditions of excitation, the duration of the after-image is fairly constant for the same person

and increases with the length of adaptation till a certain maximum is reached, but varies considerably with different persons and with the illumination of the test object.

J. E. LEBENSOHN.

Refraction and Accommodation

CHANGES IN OCULAR REFRACTION IN DIABETES. C. CASTRIGNANI, *Ann. di ottal. e clin. ocul.* 63: 869 (Nov.) 1935.

A girl of 16 years complained of a sudden loss of vision. Vision was reduced to 1/10, and myopia of 4 diopters was present. The patient was diabetic and had received some treatment previously but then had returned to a normal diet. A week later vision was 4/10, and the myopia was reduced to —3.00. Ten days later the vision was 12/10 in each eye without correction.

Castrignani reviews the various theories in explanation of such refractive changes in diabetes but believes that the problem has not been solved.

S. R. GIFFORD.

AN EPIDEMIC OF BILATERAL PARALYSIS OF ACCOMMODATION IN CHILDREN. A. FUCHS, *Wien. klin. Wchnschr.* 48: 1547 (Dec. 13) 1935.

Within a month's time after Oct. 10, 1935, Fuchs saw four girls with bilateral paralysis of accommodation. These were girls from 8 to 16 years of age. They had no complaints other than some blurring of vision, and in each case both the history and the general physical examination were unproductive of a cause. In each case the accommodation was reduced from 13 to between 3 and 4 diopters. The near point for accommodation was between 23 and 32 cm. The rarity of this condition, revealed in the fact that only one case, seen by Genet in 1934, has been reported in the literature, is the reason for this report. Such cases are found following cases of diphtheria and influenza, but because of the transient nature of the paralysis and because of the fact that oculists are not called to see the patients the cases are not often reported in the literature. Usually there is an accompanying paresis of one or more extra-ocular muscles as noted following encephalitis. The girls were not bacteria carriers, were in various parts of the country during the preceding summer and gave no history of coryza or other disease. The prognosis is at present unknown. In the postdiphtheritic type symptoms are absent after one week. A table considering the age, refraction, punctum proximum, accommodation and remarks accompanies the article.

L. L. MAYER.

Retina and Optic Nerve

THE OBLITERATION OF RETINAL TEARS BY DIATHERMOCOAGULATION. P. VEIL and M. A. DOLLFUS, *Arch. d'opht.* 52: 162 (March) 1935.

The results of one hundred operations by diathermocoagulation, the earliest dating from July 1933, are reported. In one series the method of Weve or of Šafář was used; in a second, the pyrometric technic of

L. Coppez, slightly modified. The operations were done in a dark room under ophthalmoscopic control, for which a contact glass is not indispensable. The dose of current was regulated so as to obtain the minimum reaction visible to the ophthalmoscope. Perforating diathermo-coagulation is easy to perform over a limited area. It is difficult if carried out over an extensive area. In the latter case surface coagulation is preferable. Veil and Dollfus prefer diathermic puncture for draining off the subretinal fluid rather than use of a knife. They have obtained cures in 55 per cent of cases by perforating diathermy and in 58 per cent of cases by surface coagulation with the pyrometric electrode. Cicatrization is much slower after electrocoagulation than after thermocautery or galvanocautery. Therefore the period of rest in bed should be much longer, and stenopaic glasses should be worn for at least one month.

S. B. MARLOW.

RETINAL ARTERIAL PRESSURE IN RELATION TO THE GENERAL CIRCULATION AND TO CERTAIN OCULAR DISEASES. E. MARCHESINI, *Ann. di ottal. e clin. ocul.* 63: 532 (July); 621 (Aug.) 1935.

Records were made with the Bailliant dynamometer for a series of normal and a series of diseased eyes before and after administration of acetylcholine. In twenty-one normal eyes the average systolic pressure in the retinal arteries was 73.6 mm. of mercury, and the average diastolic pressure was 34 mm. The relation of these figures to those for the general blood pressure was approximately that found by Bailliant, the retinal systolic pressure being 54.23 per cent of the general systolic pressure and the retinal diastolic pressure 41.4 per cent of the general diastolic pressure. After administration of acetylcholine a drop in pressure occurred, averaging 6.42 and 4.04 mm. for the general systolic and the general diastolic pressure, while the retinal pressures dropped 3.42 and 2.19 mm. A slight increase in vision accompanied this drop in pressure, amounting to from .33/10 to .4/10. Patients with high myopia and those with hyperopia showed no differences in response as compared with normal persons. Patients with general hypertension, including one with thrombosis of a retinal artery, showed no appreciable drop in pressure after the administration of acetylcholine and no increase in vision. In patients with optic atrophy a normal drop in pressure followed the administration of acetylcholine, but there was no improvement in vision. In three patients with retinitis pigmentosa hypotony was observed in the retinal arterial pressure as compared with the general arterial pressure. The fall of pressure obtained with acetylcholine was almost twice the normal average, and vision was increased slightly. In six patients with retinal angiospasm relative hypotony was also observed, and the fall in pressure after the administration of acetylcholine was much greater than normal. The most marked increase in vision following the use of acetylcholine was observed in this group, the average amounting to 2.16/10 for right eyes and 1.5/10 for left eyes. Five patients with retrobulbar neuritis showed slight relative hypotony, a fall of pressure after the administration of acetylcholine which was about normal and marked increase in vision, though not so marked as in the previous group.

S. R. GIFFORD.

OCCCLUSION OF THE CENTRAL RETINAL ARTERY. F. GABARDI, *Rassegna ital. d'ottal.* 4: 333 (May-June) 1935.

Gabardi examines critically the possible causes concerned in occlusion of the central retinal artery (embolism, endarteritis obliterans, thrombo-arteritis, angiospasm) and the differential diagnosis. He presents five cases of partial or total occlusion.

A clinical differentiation of the causes cannot be easily outlined or predicated from clearly pathognomic signs. A differentiation is possible but only on the basis of a personal evaluation, and on this account it cannot be absolute. This failing should be an incentive for new, more accurate observations which will carry the observer beyond the usual diagnosis of embolism.

V. R. SYRACUSE.

PERIPHLEBITIS AND ENDOVASCULITIS OF THE RETINAL VESSELS; SCLEROTIC, TUBERCULOUS AND SEPTIC DISEASES OF THE CHOROID: REPORT OF CASES. W. GILBERT, *Klin. Monatsbl. f. Augenh.* 94: 335 (March) 1935.

Gilbert refers to Marchesani's research on juvenile angiopathy of the retina, on recurring hemorrhages in the vitreous and on obliterating thrombo-angiitis as described by Buerger. He points out that Marchesani limited the frequency of tuberculosis as a cause in these cases, many of which he attributed etiologically to general diseases of another nature. He reports two of three cases in which he found tuberculosis absent but syphilis present, yet evidently other general diseases as the etiologic factors. The first case was that of a woman, aged 48, who suffered sudden failure of vision in her left eye in November 1933. Only a small hemorrhage in the retina, surrounded by an opaque area in this membrane, could be noted in the fundus. Antisyphilitic treatment failed to improve the vision. Pallor of the disk was noticed a few weeks later, simultaneous with abdominal spasms. Swelling of the left leg and then thrombosis in the right leg appeared in the latter part of January 1934. Vascular obliteration caused by endarteritis, i. e., thrombo-angiitis as described by Buerger, with consequent gangrene of the lower extremities was considered the cause of death, which occurred on Jan. 30, 1934. The disease began with obstruction of the central retinal vessel of the left eye; spasms in the abdomen followed, and vascular obliterations in the lower extremities caused the patient's death.

In the second case, that of a man aged 28, one eye presented typical perivasculitis with involvement of the retinal vein and artery, encased veins and hemorrhages in the area of the inferior temporal branch of the retinal vein. Full vision was recovered after three months. Tuberculosis was absent, but the Wassermann reaction was strongly positive. Thrombosis in the lower part of the left leg developed later and lasted for three weeks.

Both patients complained of a sensation of cold in their feet, one of the symptoms mentioned by Buerger and Marchesani.

Gilbert doubts the correctness of Marchesani's standpoint in excluding tuberculosis in all cases of this type. He refers to tubercle bacilli which he found in the walls of the veins and in their close surroundings and to giant cells.

In the second part of his paper he discusses the differential diagnosis of diseases of the choroid caused by nephritis, arteriosclerosis, tuberculosis and septic conditions. These considerations are worthy of notice but too detailed to be enumerated in an abstract. Two items may be mentioned: The first is the case of a woman, aged 63, who had severe inflammation of the anterior part of the uvea in one eye. Very firm posterior synechiae caused secondary glaucoma. After iridectomy an area of sclerosis of the choroidal vessels about 3 disk diameters in circumference was noted in the macula. Choroidal foci were absent. Similar conditions existed in the other eye. The cause of this disease, in Gilbert's opinion, was sclerosis of isolated areas of the uveal vascular system; tuberculosis, syphilis, rheumatism and infections could be ruled out.

As to disseminated choroiditis, he arrives at the conclusion that it is of tuberculous origin, and that its very presence is indicative of the tuberculous primary complex which gave rise to it.

K. L. STOLL.

Trachoma

PRELIMINARY NOTE ON THE PRESENCE OF INFRAMICROBIAL ELEMENTS IN TRACHOMA FOLLICLES. A. CUÉNOD, Arch. d'opht. 52:145 (March) 1935.

In this report the attempt is made to establish firmly the existence of intrafollicular bodies in trachoma. With few exceptions, due probably to faulty technic at the beginning of the study, these bodies have always been found in cases of active trachoma, several hundred in number. They are not only constant but exceedingly numerous and appear in two general forms—one extremely small, which corresponds to what has been called the "elementary corpuscle" in inclusions proper, and the other much larger, analogous to what has been termed the initial body. The technic employed in the search for these bodies in the trachoma follicle is described. In the absence of all other pathologic bacteria these bodies have so much in their favor that one must be careful not to attribute the disease to them. In a later work Cuénod hopes to present reasons for believing them to have an active, if not an exclusive, part in the causation of the disease.

S. B. MARLOW.

SUCCESSFUL TRANSMISSION OF TRACHOMA IN MAN. E. DELANOË, Rev. internat. du trachome 12:242 (Oct.) 1935.

The right eye of an Arab, blind since childhood, from variola, had since then occasioned no discomfort and was absolutely without signs of trachoma. His wife, however, was being treated for trachoma in a hyperacute form. A fine applicator covered with sterile cotton was well rubbed over her tarsal conjunctiva and then well rubbed on the tarsal conjunctiva of his blind eye. After a week itching, tingling and discharge affected the blind eye. Five weeks later, examination disclosed a succulent tarsal conjunctiva studded with follicles.

In two other blind eyes inoculation failed, probably because the secretion was merely applied, not thoroughly rubbed in.

J. E. LEBENSOHN.

INOCULATION OF TRACHOMATOUS MATERIAL INTO THE BRAINS OF GUINEA-PIGS. A. VON SZILY, *Klin. Monatsbl. f. Augenh.* 95: 433 (Oct.) 1935.

Referring to previous experiments in intracranial transmission of herpes, von Szily reports on a series of similar experiments with trachomatous material. Guinea-pigs were used. Under slight ether anesthesia and after shaving and disinfecting a small area of the galea of a guinea-pig the skull was prepared free; a small opening from 1 to 2 mm. in diameter was drilled through the skull, and 0.1 cc. of a weak solution of trachomatous conjunctival tissue was injected into the brain. The wound in the galea was closed with sutures. No cerebral or general physical symptoms resulted. From two to three months later the skull was opened in a manner described in detail, and the brain removed. The site of the injection was barely visible, but numerous small gray nodules, ranging in size up to that of a millet seed, were noted in the meninges, especially in the median line of the convexity of the brain. Practically no reaction in the brains was observed histologically, and perivascular accumulations of cells such as those found in encephalitis were absent. A number of photomicrographs illustrate the histologic observations. Large isolated follicles were found in the meninges but separated from the blood vessels; they occurred in the dura mater and in the arachnoid and between these membranes. The results corresponded with those obtained by intra-ocular injections, and numerous germ centers in the shape of trachoma follicles were in evidence. The nervous elements were free from follicles, as they were absent in the retina after intra-ocular injections. The distribution of the follicles over a large area is indicative of the activity of the specific noxa.

K. L. STOLL.

Tumors

A CYLINDROMATOUS BASILOMA OF THE LOWER LID. G. W. CAOCCI, *Ann. di ottal. e clin. ocul.* 63: 519 (July) 1935.

Caocci describes a basal cell carcinoma involving the lower lid, at the outer angle. The unusual feature was the presence of numerous cavities lined with epithelium. The cavities contained a substance with the staining properties of mucin. The growth apparently originated in a previously ectropionized conjunctiva, and the excessive formation of mucin was apparently responsible for the formation of cavities.

Photomicrographs and a bibliography accompany the article.

S. R. GIFFORD.

TUMOR OF THE CHOROID. M. LAIGNIER, *Bull. Soc. d'opht. de Paris*, June 1935, p. 357.

A man 35 years old consulted Laignier because of a small brown freckle on the iris of his right eye; the freckle appeared to be enlarging. The visual acuity was normal. Normal findings were recorded from the general examination. A large iridectomy including the mass was accomplished. The pathologic diagnosis was melanotic sarcoma. Four or five months later, when the fundus of the affected eye was examined,

there was noted at 6 o'clock, two disks' width from the ora serrata, a small white spot resembling an exudate. Roentgen examination and use of a magnet showed no foreign body in the eye. Transillumination indicated a large tumor. Subsequently the retina over the affected area became detached. Photographs of the fundus are shown. The patient refused removal of the eye. Laignier asks whether diathermy should be applied.

L. L. MAYER.

HEMOGEMANGIOMA OF THE UPPER EYELID WITH EXTENSION TO THE LOWER LID, TEMPORAL REGION AND ORBIT. A. NATALE, Arch. de oftal. de Buenos Aires 10:587 (Aug.) 1935.

Natale reports a case of hemogemangioma. He gives this name to the tumors formerly known as fibro-angiomas, fibrohemangiomas and fibrous hemangiomas which were under the generic name of elephantiasis, on account of their embryonic origin from successive vascular spurts.

The patient was a man 30 years of age, without personal or hereditary antecedents of this condition. A horseshoe-shaped tumor involved the upper eyelid and part of the lower eyelid as well as the temporo-frontal region. The palpebral fissure was narrowed and lower than that of the other eye. The eyeball was pushed downward and inward. The cornea was covered by a xerotic secretion; the bulbar conjunctiva was red, swollen and infiltrated above by the yellowish tumor. The consistence of the tumor was soft, elastic and non-nodular.

Natale describes the clinical characteristics of angiomas and hemangiomas, distinguishing two varieties: (1) simple or telangiectatic and (2) cavernous, the latter being circumscribed or diffuse. The present tumor belonged to this variety.

The eye was treated surgically in four sessions, which are described in detail.

The pathologic examination revealed a fibrillar interstitial connective tissue enclosing new-formed capillaries which were distributed in a disorderly manner, with some mast cells.

The literature on the subject is reviewed on the basis that tumors of this type were formerly known as elephantiasis, the present one being almost identical with that described by van Duysen in 1889.

C. E. FINLAY.

TWO CASES OF RECKLINGHAUSEN'S DISEASE. F. J. SORIANO and H. PÍCOLI, Arch. de oftal. de Buenos Aires 10:620 (Aug.) 1935.

Soriano and Pícoli report two cases of painless tumor of the eyelid. In one instance spots of pigment and papillomatous nevi were found on the skin of the back and face. In both cases histologic examination showed fibroblastic cellular elements in a nestlike arrangement, which in transverse sections had an appearance similar to that of nerve fibers. In each case the neurofibroma was located in the supra-orbital branch of the superior division of the trigeminus nerve.

The reports are preceded by a detailed account of the symptoms of Recklinghausen's disease when these differ from the classic triad and of the different ocular lesions possible. The literature on the subject is extensively reviewed.

C. E. FINLAY.

Uvea

DIAGNOSIS AND SPECIFIC TREATMENT OF TUBERCULOUS CHOROIDITIS.
A. SAMOJLOFF and A. TIHOMIROVA, *Ann. d'ocul.* 172: 113 (Dec.) 1935.

The ophthalmoscopic diagnosis of tuberculous choroiditis presents great difficulty. The authors have seen many cases in which ophthalmoscopic examination gave no indication of the cause of the choroidal disease, and it was only by successful treatment with tuberculin that the tuberculous nature of the disease was shown. The difficulty is due to the fact that the tuberculous process in the choroid in many cases does not present any specific sign. On account of these difficulties, it is the opinion of many authors that tuberculous lesions of the choroid are rare. The first cases of tuberculous choroiditis to be recognized were those found in subjects with tuberculosis, and it was in these cases that the tuberculous origin of the condition was first admitted. Also in cases of tuberculous meningitis the specific origin of choroidal infection was generally recognized. It was only at a much later date that similar choroidal lesions in cases of chronic benign tuberculosis were admitted to be due to this cause.

Systematic ophthalmoscopic examination of the eyes of thirty-three persons with tuberculous choroiditis (fifty diseased eyes) showed a focal reaction of the choroid after subcutaneous injection of tuberculin into all in which tuberculosis was present.

The article is illustrated by two charts.

S. H. McKEE.

Vitreous

THE CYTOLOGY OF THE DISEASED VITREOUS. M. LÓPEZ ENRÍQUEZ, *Arch. de oftal. hispano-am.* 35: 197 (April) 1935.

This is a preliminary report on the microscopic demonstration of the migration of retinal pigment into the vitreous in pathologic cases, with predominance of Hortega cells and with variations depending on accidental and local conditions. The author deduces from different authors' observations of the cellular element of the cerebrospinal fluid that in pathologic cases the Hortega cells can migrate into this fluid.

C. E. FINLAY.

DEGENERATIVE PROCESSES IN THE VITREOUS OF THE HUMAN EYE AND THE ORIGIN OF TEARS IN THE RETINA. G. BAURMANN, *Klin. Monatsbl. f. Augenh.* 95: 259 (Aug.) 1935.

Baurmann examined the vitreous body in situ in four enucleated eyes with an especial technic. The ages of the patients were 16, 31, 34 and 55 years, respectively. Four distinct types of degeneration of the vitreous had occurred, leading to a formation of vacuoles within the vitreous. These changes in the vitreous increased with age. The formation of vacuoles, filled with a fluid, began with a typical transformation of the normal stringy structure of the vitreous into a coarse network of granular cords. This tendency of the vitreous to degenerate rela-

tively early and the anatomic fact that the vitreous is closely connected with its surroundings only in the area of the orbiculus ciliaris and the most anteriorly located portion of the pars optica of the retina, in Baurmann's opinion, form the base for the formation of some of the retinal tears. When the liquefaction of the vitreous has reached an advanced stage the tears in the retina occur in this manner: Isolated pearls of vitreous which adhere to the anterior portion of the pars optica of the retina move contrary to the motion of the liquefied contents in the vitreous chamber when the eyeball moves; traction resulting at the insertion of the pars optica causes the tears in the retina. Applying Brückner's measurements of the velocity of ocular motions, Baurmann found that a pearl of vitreous of the bulk of 0.1 Gm. will exert a traction equivalent to 0.3 Gm.

K. L. STOLL.

Sympathetic Ophthalmia

STATISTICS ON THE OCCURRENCE OF SYMPATHETIC OPHTHALMIA. B. WALDMANN, *Arch. f. Augenh.* 109:441, 1935.

Waldmann has already called attention to his theory that sympathetic ophthalmia is of ectogenous origin and that the exciting cause is to be found in the nasal accessory sinuses. He supposes that it reaches the eye from there along the pia mater of the optic nerve or the ciliary nerves.

The present article is concerned with further proof based on statistics from a number of clinics scattered over the world. In answers to a questionnaire he finds that, of 193 cases of sympathetic ophthalmia observed in these clinics, 146 occurred between October and April and only 47 between May and September. He interprets this as further evidence that the cause of sympathetic ophthalmia is associated with infections of the upper respiratory tract, since in these months such infections are more common.

The frequent occurrence of neuritis with the beginning iridocyclitis proves that the optic nerve plays a significant rôle in the dissemination of the infectious agent.

F. H. ADLER.

Therapeutics

PRACTICAL TREATMENT OF OCULAR TUBERCULOSIS. GÓMEZ-MÁRQUEZ, *Arch. de oftal. hispano-am.* 35:177 (April) 1935.

Gómez-Márquez considers practically the whole community as being infected with tuberculosis, primarily in the first months of life, and as reacting in the presence of reinfections according to the degree of immunity and allergy developed.

In cases of ocular reinfection of the iris, for example, high immunity with high allergy produces acute iritis with complete recovery; high immunity with low allergy, mild iritis with complete recovery; low immunity with high allergy, acute iritis with subsequent tubercle formation in the iris; low immunity with low allergy, mild iritis with tubercle formation in the iris. It is therefore essential, especially in the treatment of ocular tuberculosis, to determine primarily what elements of the

clinical picture depend, respectively, on the immunity and the allergic factors, and then to attack the germ, first directly and second indirectly, and finally to modify the existing allergic condition. For the first, the author relies on chemotherapy, preferring the gold salts, especially sanocrysin. For the second he uses the old treatment with iodine, arsenic and iron and change of climatic environment—the seaside for torpid conditions and moderate altitudes for crethitic and congenital ones, with prudent aeration and sunlight. For the third group he recommends a combination of these two forms of treatment and the administration of tuberculin if the lesions point to hypersensibility.

He further divides tuberculous lesions into three groups: (1) destructive lesions produced by metabolic action of the germ, without congestive exudative phenomena of tubercle production, in which chemotherapy and hygiene are indicated; (2) congestive and exudative lesions with tubercle production, due to hypersensibility, in which tuberculin should be used; (3) mixed lesions, in the treatment of which both procedures should be combined.

C. E. FINLAY.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Feb. 17, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

METHYLTHIONINE CHLORIDE (METHYLENE BLUE) STAINING OF THE EYE. DR. ROBERT K. LAMBERT.

Three patients presented an interesting and unusual appearance of the ocular fundus. On ophthalmoscopic examination, the fundus appeared as though seen through a blue filter. The patients had been receiving methylthionine chloride U. S. P. (methylene blue) by mouth over a long period. The eye of one of the patients was examined after death, and it was found that the vitreous had taken a blue stain.

DISCUSSION

MR. E. B. BURCHELL: When the eyeball was opened, the vitreous showed a much deeper shade of blue than it does at present, and as the process of dehydration went along the blue faded. The eye had been fixed in Bouin's solution, which consists mostly of tri-nitrophenol, and it is a question whether the entrance of the tri-nitrophenol into the globe was not the cause of the loss of some of the blue, as after the eye had been sectioned and mounted microscopic examinations revealed no blue pigment.

DR. ROBERT K. LAMBERT: When methylthionine chloride is injected into the body, it is largely reduced to a colorless form, but in some tissues it remains blue. It seems possible that the region around the retina, having such a high oxidative rate, kept the blue form to a greater extent. My co-workers and I had called to our attention the case of a man who died from carbon monoxide poisoning and who had received methylthionine chloride intravenously. The man's brain was colorless. When it was sectioned, however, and the cut surfaces exposed to the air, these rapidly turned blue as the methylthionine chloride was oxidized. We have no explanation to offer as to why the myelin fibers of that particular specimen did not turn blue.

HOMOKERATOPLASTY. DR. JAMES G. MORRISSEY.

Complete homokeratoplasty was done on a young woman of 23 who had a painful leukoma that had developed after keratomalacia at the age of 2 years.

THE VALUE OF THE CONTACT GLASS IN THE THERAPY OF LAGOPHTHALMIC AND NEUROPARALYTIC KERATITIS. DR. DANIEL M. ROLETT.

The contact glass as a therapeutic agent was tried with success on two patients with corneal ulcers. In one patient a serpiginous ulcer healed within ten days after he began wearing the contact glass filled with a strong protein silver in a concentration of from 3 to 5 per cent.

The second patient had a typical ulcer of the cornea due to lagophthalmos and neuroparalytic keratitis in which the usual conservative treatment was of no avail. With the contact glass filled with physiologic solution of sodium chloride and worn for various periods during the day and night, the ulcer cleared entirely within several weeks.

PATHOLOGIC ASPECTS OF CHRONIC INFLAMMATORY CONDITIONS OF THE CORNEA. DR. BERNARD SAMUELS.

By way of preparing the audience for a better understanding of the paper that Dr. Jacob Jacobson was to read, Dr. Samuels presented a number of microscopic preparations, which were of two types:

1. Lesions that Dr. Jacobson declined to treat, quiescent old non-vascular corneal scars, some of which showed degenerative changes in the way of calcareous deposits. In declining to attempt to treat these scars Dr. Jacobson explained that the nonvascular fibrous elements of which they consisted were not amenable to absorption.

2. The opacities accepted for treatment. These consisted of vascularized subacute infiltrations lying between the lamellae of the cornea, as in interstitial keratitis of syphilitic or tuberculous origin, and of pannus, especially trachomatous pannus.

RESULTS OBTAINED WITH BENZYL CINNAMATE IN THE TREATMENT OF TRACHOMA AND CORNEAL OPACITIES. DR. JACOB JACOBSON, Paris, France.

This article will appear in full in a later issue of the ARCHIVES.

March 16, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

THE YOUNG OPERATION FOR PTOSIS. DR. WILLIS S. KNIGHTON.

The Young operation for congenital ptosis of the right upper lid was performed on a young boy whose left eye was normal. The right superior rectus muscle was normal, but the levator palpebrae was paralyzed. An adhesion was made between the superior rectus muscle and the tarsus, forming a symblepharon, according to the technic of George Young. The ultimate result was good cosmetically and functionally.

DISCUSSION

DR. W. GUERNSEY FREY: I should like to ask whether there is any interference with lateral movements of the eyeball after this operation.

DR. SIGMUND A. AGATSTON: After hearing Dr. Knighton describe the operation, I tried it on a patient two and a half weeks ago. The result so far looks very good.

DR. WILLIS S. KNIGHTON: In reply to Dr. Frey, as far as one can see, there is no interference with lateral movements of the eyeball. If one observes the patient carefully one may notice a little twitching of the eyelid when the eye turns right or left, but the patient does not complain of any interference.

BILATERAL VITREOUS CYST. DR. CHARLES A. PERERA.

Twenty instances of a unilateral cystic structure in the vitreous have been reported, but no one has had the opportunity of studying such material histologically. The origins of these bodies have been more or less speculative, although in eight of the twenty-four cases that have been recorded the cysts appeared to be definitely related to persisting congenital structures. The possible derivations of the cysts include: the fetal ocular cleft, mesodermal elements of the embryonic vitreous, the hyaloid apparatus, a cyst of the ciliary process, formed exudate in the vitreous, products of retinal degeneration or proliferation, and degeneration of a parasitic cyst.

ORBITAL TUMORS. DR. C. S. O'BRIEN.

This paper will be published in full, with the discussion, in a later issue of the ARCHIVES.

April 20, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

SIMPLE CONJUNCTIVAL SUTURE FOR USE IN SECTION FOR REMOVAL OF A CATARACT. DR. S. A. AGATSTON.

A suture to safeguard the eye in the course of operation should be simple and should be capable of being introduced with ease and speed and fastened with dispatch. A small V-shaped conjunctival flap is made, and the needles, 2 mm. apart, are passed through the flap and then through the conjunctiva above. The suture is not pulled tight, a lateral loop being left on each side, out of the way of the incision. Immediately after delivery of the lens, the assistant effects a closure in purse-string fashion by traction on the two parallel threads so that the flap slides under the conjunctiva above. Mere traction closes the wound safely, and the suture can be tied at the surgeon's leisure.

This method has been used during the past three years in all such operations on my private and public patients and by Dr. Bernard Samuels in his service at the New York Eye and Ear Infirmary.

DISCUSSION

DR. EUGENE BLAKE, New Haven, Conn.: It seems to me that failure to put the suture through the episcleral tissue sacrifices support and security.

DR. SIGMUND A. AGATSTON: I do not think it is necessary to pierce the episclera, because with the lower flap sliding under the upper the suture is just as strong as though the needle went into the episclera.

TWO TUMORS OF THE EYE: (1) AMPUTATION NEUROMA OF THE LONG POSTERIOR CILIARY NERVE. (2) PLEXIFORM NEUROMA OF THE CHOROID IN A NONBUPHTHALMIC EYE. DR. LOUISE H. MEEKER.

1. The amputation neuroma is in an eye that shows "phthisis bulbi": The patient, a 19 year old boy, has been blind since birth—an instrumental delivery. All the tunics of the eye were ruptured near the optic nerve. The nerve is enlarged with ganglions at several points. The long posterior ciliary nerve of the other side forms an intrascleral loop beneath the conjunctiva, indicating a congenital tendency toward growth. This tumor links the so-called neurofibroma of Axenfeld with that of Kyrieleis and duplicates amputation neuromas of other peripheral nerves. There are no associated evidences of Recklinghausen's neurofibromatosis.

2. The plexiform neuroma is in an eye removed for corneal ulcer and hypopyon with glaucoma. The patient, a woman, aged 41, gave a history of iridocyclitis of two years' duration which followed a blow on the eye. The eye is of normal size. The filtration angle is blocked by the adherent iris, and the disk is excavated. The choroid is thickened posterior to the equator, and the normal stroma is partly replaced by nerve fibers, which are greatly increased in number about the posterior ciliary vessels. Ganglion cells are very numerous, especially a few millimeters from the papilla. This finding in a nonbuphtthalmic eye has been reported once (Freeman). The patient's other eye is apparently normal, and there are no further evidences of Recklinghausen's neurofibromatosis.

LESIONS OF THE OCULAR FUNDUS IN PATIENTS WITH ESSENTIAL HYPERTENSION AND ARTERIAL AND RENAL DISEASE. DR. MARTIN COHEN.

This paper may be presented in summary as follows:

1. The clinical symptoms of these patients are correlated with various lesions of the fundus.

2. The lesions of the fundus in some of the patients with essential hypertension simulated those seen in the patients with nephritis, arterial sclerosis, toxemia of pregnancy and diabetes.

3. In the patients with malignant essential hypertension, however, the lesions of the fundus were more severe than those found in the patients with the other diseases considered, with the exception of those with nephritis of certain types. These lesions consisted mainly of marked venous stasis, arterial contraction, profuse hemorrhages, throm-

bores, white plaques, macular changes and edema of the optic disk. These findings were accompanied by normal or practically normal renal function, differentiating malignant essential hypertension from nephritis in a clinical sense.

4. Hypertension was noted as a common feature in all of these patients; accordingly, it was difficult, if not impossible, to state from the observations in the fundus which was primary.

5. Edema of the optic disk in association with malignant essential hypertension is significant of its end-stage, which terminates in nephritis, causing a grave prognosis regarding the life of the patient.

6. It is difficult to decide from the lesions of the fundus in the patients with essential hypertension when the malignant phase first appeared and what its existing stage is.

7. There may be a similarity between the picture of the fundus in the terminal stage of malignant essential hypertension and that observed in chronic glomerulonephritis.

8. Further microscopic studies of the retinal and choroidal vessels are necessary in order to keep abreast with the more recent investigations of the arterioles of the kidneys and other organs.

9. The differing opinions among clinicians regarding the concept of essential hypertension cause various interpretations of the pictures of the fundus. A uniform clinical concept would establish more firmly the correlation of these pictures with the observations made by internists and pathologists.

10. The number of cases presented is too small to allow one to draw definite conclusions, and the report is presented merely to stimulate further studies in this important field.

DISCUSSION

DR. HERMAN O. MOSENTHAL: Those interested in internal medicine are just beginning to appreciate the vast significance of the pathology of the blood vessels. About twenty years ago, when arteriosclerosis, thrombo-angiitis obliterans, Raynaud's disease and periarteritis nodosa were regarded as almost the only diseases of the blood vessels, the situation was static because the vascular tree was looked on largely as a system of inert tubing rather than one composed of vital tissue with a special function, subject to somewhat the same diseases that occur in the lungs, the kidneys, the gastro-intestinal tract and elsewhere. Many vascular diseases have been found since that time and are still being studied, and when Dr. Cohen says he wishes that the clinician would give him a well conceived uniform description of what the vascular changes in hypertension are, he can only be furnished with an inconclusive partial statement. It was less than twenty-five years ago that Sir Clifford Allbutt in England and Theodore Janeway in America distinguished between senile, i. e., decreascent, arteriosclerosis and hypertensive arterial degeneration; since then the necrotizing lesions of the arteries, first discovered in the renal vessels by Fahr, have been recognized as something apart. All these arterial changes occur commonly in patients with hypertension, and it is easy to see how difficult it may be to get a uniform picture of the retina since it is dependent on the

many and variable vascular changes accompanying permanently increased arterial pressure.

The so-called benign and the malignant hypertension deserve mention because these terms have been used very freely and not, I think, with thorough understanding of what they mean. The original idea of Keith and his associates was that malignant hypertension is a condition associated with increased blood pressure, retinal changes and normal renal function, with death occurring from a cerebral accident. This condition, of course, is entirely different from malignant nephrosclerosis or necrotizing arteriolitis; the term "malignant hypertension" should be applied only to this devastating change in the arteries, and not to every case of markedly high blood pressure, as is the habit of many physicians. A much greater insight into the diagnosis of the retinal lesions can be obtained if one can accurately correlate the ophthalmoscopic picture with the changes that occur in the blood vessels rather than with a clinical syndrome, such as diabetes, hypertension or glomerular nephritis.

DR. W. W. HERRICK: If I have any suggestion to make it is that a study of this sort be extended to include the earlier stages of this condition. Those present have in this study heard of the fundus changes characteristic of the late or terminal stages of arterial disease, of diabetes and of nephritis but nothing about the changes which mark the earlier stages of such diseases. I believe this is a great field for cooperative work among the internists and ophthalmologists.

Roughly, for clinical purposes, one can classify the diseases of the kidney and the arteries under discussion as the nephroses, the nephritides and the primary arterial diseases. Of the nephroses the most common is that of pregnancy. In that condition one sees spasticity of the arteries, a narrowing in their caliber, which often varies from day to day. Soon thereafter there is edema of the general retinal field, perhaps including the optic nerve head. Later one sometimes sees hemorrhage, very rarely patches of exudate and occasionally retinal separation, which, in our experience at the Sloane Hospital for Women, has a fairly good prognosis. Conditions of that sort, usually accompanied by hypertension, clear up with delivery, but in the subsequent years at least 40 per cent of the patients will come to have essential hypertension and the ophthalmoscopic changes associated with that disease. In essential hypertension, which internists consider a primary functional disorder characterized by a rise in blood pressure with secondary changes in the small vessels, with a high systolic and a correspondingly high diastolic pressure, with death resulting most often from cerebral or cardiac changes and rarely from renal insufficiency secondary to renal sclerosis—in this most common of arterial diseases, there is first of all a spastic condition of the retinal vessels: an arteriovenous constriction and distention of the veins. Only when the condition is more advanced does one see an exudate. This is most likely to be of the medium-sized grayish patch type and more rarely of the silver-white dot type. In the later stages there is hemorrhage. That is about as far as essential hypertension in the average patient goes in retinal changes unless there is reduction of the renal circulation to the point of renal failure. Then one sees papilledema, large amounts of yellowish exudate, most often in the macular region, with large hemorrhages and sometimes thrombosis. These are also the changes which one finds in patients with malignant

hypertension, which practically always ends in renal failure. It may be that this is a separate disease, since its pathology, as described by Dr. Mosenthal, is quite different and distinct from that of benign essential hypertension. In malignant hypertension the changes are those of the most advanced arteriosclerosis plus papilledema and large masses of exudate.

I believe one should confine the term "nephritis" to the inflammatory processes in the kidney; that one should not include in the term the nephropathies secondary to arterial changes. One should make an attempt to differentiate primary vascular renal disease from primary inflammatory renal disease. One cannot always do that in the late stages of either process, but in the earlier phases, and with the knowledge of the history of the disease, it is often possible. It is of great interest that the retinal changes in patients with nephritis do not run parallel with the clinical picture. One may see advanced retinal changes with moderate nephritis. One sometimes sees uremic death with very little change in the retina. I have lately observed a case in my own ward at the Presbyterian Hospital, an example of embolic nephritis with uremic death and practically no ophthalmoscopic lesions. When one does encounter retinal changes with nephritis, they are apt to be dramatic—marked papilledema, great patches of yellowish-white exudate and hemorrhage, with a secondary and relatively late development of arteriosclerosis.

DR. A. J. BEDELL: It seems to me that until one has the correlation of photographic reproductions of the fundus clinical findings and pathologic observations one will continue to indulge in speculation. Nothing but photographs of the fundus will serve the ophthalmologist's purpose.

There is a widespread misunderstanding regarding hypertension and arteriosclerosis. Dr. Cohen, fortunately, stresses one phase of the latter, and I agree with him that practically all the changes that one finds in the fundus of a patient with diabetes are arteriosclerotic in origin, with the possible exception of the hard whitish-yellow granular exudates which appear in and about the macular region. This is substantiated in bedside practice, in which, with the exception of lipemia, no changes are ever found in the fundi of children with diabetes.

Dr. Cohen makes the further statement that it is difficult or impossible for the ophthalmologist to diagnose renal or arterial disease without the cooperation of the internist. This, I am sure, is good teaching and often proved.

DR. DAVID WEXLER: I am inclined to agree with Dr. Mosenthal that the distinction between the benign and the malignant phase of essential hypertension is largely a matter of definition. The difference is an anatomic one and depends on the speed and degree to which the arterioles are narrowed. For instance, one is tempted to apply the term "malignant" to that form of essential hypertension in which a person in his thirties or early forties, usually with compensated renal function, succumbs to a cerebral or cardiac accident. Strictly speaking, malignant hypertension is synonymous with arteriolonecrosis or malignant sclerosis of the kidneys. On the other hand, the benign form exhibits varying degrees of slowly progressive arteriosclerosis of the kidneys. In this form, renal function is adequate, except perhaps in the terminal stages.

In the fundus, perhaps the safest single criterion of malignant hypertension is papilledema; furthermore, it is proper to consider the hypertension with papilledema malignant in the presence of adequate or compensated renal function, as pointed out by Keith and Wagner and by Fishberg and Oppenheimer several years ago. A clinicopathologic study of twenty-nine patients with essential hypertension observed at the Mount Sinai Hospital together with Dr. I. Goldstein confirms this opinion. This group includes only those whose eyes were studied histologically. Sixteen had benign and thirteen malignant hypertension. In the group with the benign form, death in six was due directly or indirectly to cardiac failure; three died of cerebral hemorrhage, two of arteriosclerosis of the kidneys and the remainder of intercurrent disease (cancer and various postoperative complications). In all the patients with the malignant form who were available for study, death was attributable primarily to renal insufficiency, and the kidneys showed malignant nephrosclerosis. In the group with the benign form the optic disk was invariably normal, although in nine there was retinitis of some degree. Papilledema was a constant feature in those with malignant nephrosclerosis. It is interesting to note that all of those who suffered cerebral accidents who were studied were in the group with benign hypertension. In these, the kidneys showed slight arteriosclerosis and the disks were normal in the presence of moderately severe retinitis. There was no instance of a fatal termination due to a cerebral hemorrhage in the group with malignant nephrosclerosis.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Annual Congress, London, England, April 2, 3 and 4, 1936

R. FOSTER MOORE, F.R.C.S., *President*

OPHTHALMIA NEONATORUM. MR. J. D. MAGOR CARDELL.

The commonest source of infection is in the infected material implanted as the baby's head traverses the birth canal of the infected mother. The mother or the attendant may easily infect the baby's eyes with soiled hands. In making an examination of the infant, the non-infected eye should be examined first. The purulent discharge may contain micro-organisms, which should always be sought in smear and culture. When the discharge is confined behind swollen lids, there is an adverse effect on the cornea; if excessive force is used in opening the lids, the cornea may be damaged, and if the lids are carelessly opened, the pus may spurt into the eyes of the attendant.

With regard to complications, corneal ulceration is most to be feared, and it should be remembered that the cornea is susceptible to slight trauma. The lacrimal gland is rarely involved. The duration of the disease depends on the severity of the infection, the health and degree of resistance of the child and the efficiency of the treatment. The prognosis is more favorable when the infection is slight and when

institutional treatment can be secured. It is less favorable in the presence of marasmus, prematurity, gastro-enteritis and syphilis.

The treatment carried out for the condition should consist in hourly irrigation, day and night, accomplished by two nurses, one to apply the treatment and manipulate the upper lid, the other to steady the child's body and to retract the lower lid. Drops used are silver nitrate, strong protein silver, mild protein silver, mercurochrome, acriflavine, liquid petrolatum, hydrogen peroxide and lactic acid.

DISCUSSION

DR. H. P. NEWSHOLME: Most people think of gonorrhea when ophthalmia neonatorum is mentioned, but bacteriologic methods inculcate other organisms beside the gonococcus. The medical practitioner is required to notify the local authority of infants with purulent discharge beginning within twenty-one days after birth. Though the rates of incidence vary, owing to the varying degrees of energy shown by those engaged in notification, there has been in Birmingham in the last few years a marked diminution in the number of cases of severely impaired vision. The decrease of gonorrheal ophthalmia in Birmingham may be the result of three main factors: (1) a decrease in the prevalence of gonorrhea, (2) more efficient treatment and (3) an improved midwifery service, with better antepartum and better natal care.

With regard to prevention, the more freely and accurately pregnant women are examined the better. The Central Midwives Board has insisted on regular antepartum care and prompt reference of any abnormality to practitioners for advice. A 1 per cent solution of silver nitrate is instilled by the midwife into the conjunctival sac after birth, and if there is a conjunctival discharge the case must be reported to the local authority.

DR. S. H. BROWNING: The diagnosis and treatment in any given case must depend on the bacteriologic observations. Without the aid of bacteriology even the most skilful clinician must experience diagnostic difficulty. Treatment by vaccines and serums is not satisfactory. In patients with bilateral involvement both eyes tend to be cured at the same time, thus appearing to suggest that the level of resistance of the body is raised or that local immunity has occurred in both eyes at the same time. The prospect of cure depends largely on the constitution of the child. Syphilitic children with ophthalmia neonatorum do badly. Only 48 per cent of syphilitic children recovered without loss of sight, while 81 per cent of nonsyphilitic children recovered without such loss. Recently, *Pneumococcus* has frequently been found associated with ophthalmia neonatorum.

PROF. M. MÁRQUEZ: Means of fighting ophthalmia neonatorum are often used to excess. The use of silver nitrate is the standard medication, but the use of the silver nitrate stick is to be strongly deprecated. Potassium permanganate, 1 part in 4,000, used twice or thrice daily, is valuable. I am not convinced as to the value of strong protein silver (protargol and similar preparations).

SIR JOHN PARSONS: I consider that statistics based on notification returns are practically valueless, because the returns report many condi-

tions which are not simple and cannot be placed under one heading. It would be a different matter if separate tables could be drawn up of the cases of known gonorrheal ophthalmia neonatorum of the benign form.

DR. A. J. BALLANTYNE, Glasgow: There is no noticeable diminution in the number of cases of ophthalmia neonatorum in the Glasgow returns, but in the average case the severity of the condition is very much less than formerly.

COLONEL HENRY SMITH: The one method of dealing with the infected conjunctival sac is to divide the outer canthus freely and allow free drainage. There is no risk of injuring the cornea.

MR. A. F. MCCALLAN: In Egypt gonococcic ophthalmia is very prevalent, but ophthalmia neonatorum is very rare. The reason for this has never been given. It may be that trachoma and pannus with its vascularization protect the cornea.

DR. SPENCE MEIGHAN: I wish to emphasize the need for repeated bacteriologic examinations if no gonococci are found at the first examination.

MR. FRANK JULER: In a large maternity hospital, among 44,357 live babies delivered, 180 had ophthalmia neonatorum, an incidence of 0.47 per cent. A considerable proportion of the mothers were very poor, and some of them were unmarried.

MR. CHARLES GOULDEN: It is often taught that if an eye with this disease comes under treatment with the cornea intact and the eye is then lost it must be the fault of the person treating the eye. I have always held that streptococcic infection is an exception to this generality, because whatever one does in cases of streptococcic ophthalmia the eye frequently is lost. When using silver nitrate on the eyes of a baby, it is important to neutralize the silver nitrate with sodium chloride, because the baby has no tears and so there is no natural admixture with sodium chloride such as one would have in the eye of an adult.

A NEOPLASM OF THE TEMPORAL FOSSA ASSOCIATED WITH PROPTOSIS OF THE CORRESPONDING EYE. SIR ARNOLD LAWSON.

A woman aged 54 shows a growth occupying the right temporal fossa, with proptosis of the right globe. About 1924, she noticed a fulness over the right temple. Examination shows a large elastic uniformly smooth tender swelling occupying the whole of the temporal region, without glandular enlargement, discoloration of the skin, enlargement of superficial vessels or sign of inflammation. There is no limitation of movement or diplopia. The pupils are equal, small and sluggish to light stimulus, and the fundus and the circulation are normal. The thyroid gland is not enlarged. The late Sir Anthony Bowlby thought that it was probably an inoperable fibrosarcoma involving the right orbit and recommended treatment with radium, which was given in the Radium Institute. There is a diffuse thickening of the right side of the skull, the sclerosis involving the bony orbit. Forty milligrams of radium element screened with 2 mm. of lead was applied to the region of the tumor on a number of occasions, extending over two years. The growth became softer and less prominent, but in the last two years there has been little change. It seems that the exophthalmos and the temporal

growth occurred as a curious coincidence, that they were not related to each other. The thyroid now seems to be more conspicuous than formerly. The visual field is normal, and the central color vision is excellent. The growth is evidently of a low type of malignancy.

EXPERIMENTAL HETEROGENEOUS CORNEAL GRAFTS. MR. J. W. TUDOR THOMAS.

This is an account of six experimental corneal grafts on rabbits, the transplanted tissue being obtained from the corneas of animals of another species. One of the grafts was taken from a human cornea and the other five from corneas of cats. In each case use was made of a graft having a shelving margin. Unlike successful homogeneous grafts, which remain transparent, heterogeneous grafts become vascularized. The blood vessels come from the iris or, by growth through the recipient cornea, from the limbus. The primary union of the graft tends to be incomplete, and some loss of tissues from the graft readily occurs. The epithelium is lost and is replaced by a growth of epithelium from the recipient cornea, commencing in much less than two months. The epithelium is supported by a subepithelial layer of new vascularized tissue, derived largely from the stroma of the recipient cornea. Behind the graft a layer of fibers is formed from the endothelium and deep corneal fibers of the recipient eye, and behind this there develops a new posterior elastic membrane which is less than one third of the thickness of the Descemet membrane of the recipient cornea. While the original epithelium of the graft is lost, Descemet's membrane remains. The original stroma of the graft can still be distinguished as isolated fibers in the subepithelial level, but in the posterior level it remains segregated, by the growth of new tissue around it, as a mass of poorly staining fibers, fairly homogeneous in appearance, with few nuclei and practically no blood vessels. These observations seem to indicate that the heterogeneous graft is tolerated but largely ostracized by the recipient eye and that therefore heterogeneous corneal grafts should not be used for corneal transplantation in man.

RADIUM NECROSIS OF THE CORNEA. MRS. PHILIPPA MARTIN.

The time of onset of corneal necrosis depends on the intensity of the irradiation. The earliest sign is a diminution of corneal sensation, the response to touch with cotton-wool being slower than in the normal eye. After a few weeks or months the cornea may lose its polish, and later small superficial ulcers may appear and coalesce to form a large superficial ulcer. Pain becomes severe when there is a punctate infiltration, and with true irradiation iritis the pain is severe. In all cases in which massive irradiation is proposed, the pupil should be dilated with a mydriatic before the treatment is commenced and its exact size noted. After irradiation the degree to which the pupil dilates under the same mydriatic is then an indication of the presence or absence of iritis. Protection of the eye by a metallic screen is dangerous, since the screen becomes a source of secondary irradiation.

Book Reviews

Detachment of the Retina; Operative Technique and Treatment. By J. Cole Marshall. Price, \$2.75. Pp. 88. London: Oxford University Press, 1936.

This small book does not pretend to discuss the subject of retinal detachment completely. There is no consideration of the pathogenesis or even a description of the various clinical types of detachment. Marshall is wholly concerned with the treatment of detachment by modern operative methods. Four types of procedure are described—the cautery puncture of Gonin, the chemical method of Guist and of Lindner, the method of electrocoagulation of Weve and of Šafář and the use of electrolysis by Vogt and by von Szily. The descriptions include practical details observed by the author in most of the clinics where the procedures were first employed and details noted by him in his own operations.

Preparation for the operation is properly emphasized, the most important part of which is localization of the retinal tear that is usually present. This is mapped out in relation to various landmarks, and its position is recorded on a chart for use in the operating room. The charts published by Hamblin for this purpose, which seem convenient, are shown in illustrations.

Marshall feels that the procedure of Gonin, which most ophthalmologists have abandoned in favor of methods of electrocoagulation, possesses merits which will cause it to be used more frequently in certain cases, especially those in which there is a single small hole. He describes the combination of this method with scleral coagulation, which was employed by Gonin in his later operations. He seems to have had more experience with the method of Weve and Šafář, in which are employed both surface coagulation to the sclera and pins 1.5 mm. long which are inserted in the area already coagulated and with which the choroid in the area of the tear is coagulated. The importance of repeated observation of the fundus during the operation, to enable one to be sure of the relation of the coagulated areas to the tear, is emphasized. Weve's longer needles are employed for this purpose, when necessary. The illustrations showing the results of operation in illustrative cases are excellent.

In discussing electrolysis Marshall describes both the method employed by Vogt and that employed by von Szily, but has apparently failed to grasp von Szily's careful explanation of the rationale of his procedure. The method employed by von Szily depends on the application of both poles of the circuit to the eye at a short distance apart, so that the whole effect is confined to this small area. The effect is not that of heat, as with the methods of diathermy, but is a chemical effect due to the dissociation of hydrogen and hydroxyl ions in the tissues. Either both the poles are applied to the sclera or one is placed on the sclera while the other penetrates to the choroid. From the short

bibliography one misses some of the more important articles by the authors whose works were consulted. One misses also a studied comparison between the methods, especially as to the percentage of the successful results obtained with each, and conclusions as to the indications for the various methods in certain types of detachment. No summary of the author's own results is given.

SANFORD R. GIFFORD.

La radiographie en ophtalmologie. By E. Hartmann. Price, 120 francs. Pp. 280, with 391 illustrations. Paris: Masson & Cie, 1936.

This is the eighth of a series of works published under the auspices of the French Ophthalmologic Society. In format, quality of paper, reproduction of the numerous plates and in contents it comes up to the standards set by this series, which has provided a notable addition to the ophthalmologic literature of the past ten years. The illustrative roentgenograms were chosen from Hartmann's collection and from a large number sent by colleagues of various countries in response to his request.

The various methods of exposure are considered with regard to their advantages in demonstrating the parts of the orbit, with illustrative roentgenograms. By the method of Brunetti, for instance, which seems to be little known in America, perfectly clear outlines of both sphenoidal fissures are shown on one plate. This is achieved by placing the patient's brow on the plate, while the tube is employed at an angle of 15 degrees to the right and left sides, the opposite half of the plate being protected by lead during the exposures.

The visibility of calcification in the lens, vitreous and choroid is described and illustrated. Calcification of the cerebral vessels in the region of angiomas associated with von Hippel's disease is illustrated, and numerous illustrations of calcification of the internal carotid and ophthalmic arteries with evidence of pressure on the optic nerve are shown.

The detection and localization of intra-ocular foreign bodies are fully discussed. It is worth remembering that after removal of a metallic foreign body a sufficient deposit of metallic salts may be left to cast a definite shadow suggesting that a foreign body is still present. Fragments of wood and glass are illustrated by views taken under favorable conditions. The procedure of Wilder, who placed a piece of the same glass, in cases in which it was available, on the cheek to determine whether it was radiopaque, might well have been mentioned. Six groups of methods employed for localization are discussed, including the "bone-free" method of Vogt, the geometric methods, among which that of Sweet is best known, and the methods depending on making the globe visible by the injection of a radiopaque substance into Tenon's capsule. Of the "simple methods," the use of a contact glass with a radiopaque mark corresponding to the limbus, according to the method of Wessely, and the method of Velter, in which beads of lead are fastened to the limbus above and below, are among the most practicable. The latter method is almost the same as that of H. Gifford, who employed pieces of silver wire in the same way.

A great variety of tumors of the orbit are illustrated, as are a variety of fractures involving the orbit and the optic foramina. Thirty different methods of exhibiting the optic foramen have been proposed, and these, including the author's own method, are discussed.

Roentgenography of the lacrimal passages with the aid of opaque fluids is discussed and illustrated, as are the bony changes in cases of sinus disease, cerebral tumors, tumors of the hypophysis and intracranial hypertension.

The book should greatly aid both the ophthalmologist and the roentgenologist in obtaining the most useful views and in properly interpreting them.

SANFORD R. GIFFORD.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ost-flandern, Belgium.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: December 1937.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Oxford. Time: July 22-24, 1936.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. R. Foster Moore, 53, Harley St., London, 1.
Secretary: Miss Ida Mann, 7, Wimpole St., London, 1.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: July 6-8, 1936.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.

Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.

Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. John Green, 3720 Washington Blvd., St. Louis.

Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Place: Kansas City, Mo.: Time: May 11-15, 1936.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: New York. Time: Sept. 26 to Oct. 3, 1936.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. L. S. Greene, 1710 Rhode Island Ave., N. W., Washington, D. C.

Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

Place: Hot Springs, Va. Time: June 1-3, 1936.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.

Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.

Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.

Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.

Place: Salt Lake City, Utah. Time: Spring, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.

Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

Place: Seattle. Time: January 1937.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. R. Fringer, 228 S. Main St., Rockford, Ill.

Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.

Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
 Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
 Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
 Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
 Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
 Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
 Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
 Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
 Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
 Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

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President: Dr. Walter L. Hogan, 750 Main St., Hartford.
 Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
 Time: May, November.

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President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
 Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.
 Place: Savannah. Time: May 12-15, 1936.

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 Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

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 Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
 Place: Marshalltown. Time: September 1936.

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 AND OTOLARYNGOLOGY

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Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.

Place: St. Paul. Time: May 1936.

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Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

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President: Dr. Burton W. Fassett, Geer Bldg., Durham.

Secretary-Treasurer: Dr. Casper W. Jennings, 332 N. Elm St., Greensboro.

Place: Durham. Time: October 1936.

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President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.

Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

Place: Jamestown. Time: May 1936.

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President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.

Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.

Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

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Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.

Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.

Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

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Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

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Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.

Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
 Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
 Place: Richmond. Time: May 1936.

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 AND THROAT SECTION

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 ON EYE, EAR, NOSE AND THROAT

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 Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of
 each month, October to May.

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President: Dr. William O. Martin Jr., 478 Peachtree St., Atlanta, Ga.
 Secretary: Dr. A. V. Hallum, 478 Peachtree St., Atlanta, Ga.
 Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each
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 Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m.,
 fourth Thursday of each month from October to May.

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 Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
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 Thursday in February, April, May, October and December.

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 Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.
 Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
 Place: Mountain City Club. Time: Second Thursday of each month from
 September to May.

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 Monday of each month from October to May.

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Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

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AND OTOLARYNGOLOGY

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Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.

Place: Winton Hotel. Time: Fourth Friday of each month.

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Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

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Place: Athletic Club. Time: First Monday of each month.

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Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.

Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

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Time: 7:45 p. m., third Monday of every month, September to May.

DETROIT OPHTHALMOLOGICAL CLUB

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Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

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Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.

Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.

Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.

Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

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 Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

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 Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

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 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

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 Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
 Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

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 Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
 Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William A. Boyce, 727 W. 7th St., Los Angeles.
 Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
 Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

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President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
 Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
 Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

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 Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
 Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
 Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
 Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

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 Place: University Club. Time: 6 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

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 Secretary: Dr. C. W. Rucker, 527 Medical Arts Bldg., Minneapolis.
 Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

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 Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
 Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

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 Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
 Time: Second Thursday of October, December, February and April.

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 Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

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President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
 Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
 Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

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 Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St., New York.
 Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

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OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
 Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
 Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

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 Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
 Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

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 Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
 Time: First Thursday of each month from October to May.

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 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

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President: Dr. Robert H. Courtney, Professional Bldg., Richmond, Va.
 Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
 Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

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 Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
 Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

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 Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
 Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-Oto-LARYNGOLOGICAL SOCIETY

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
 Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
 Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
 EAR, NOSE AND THROAT

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
 Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
 Place: Society's Building, 2180 Washington St., San Francisco.
 Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
 Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
 Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

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President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
 Secretary: Dr. Philip B. Greene, Old National Bank Bldg., Spokane, Wash.
 Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

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Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto.

Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.

Time: First Monday, November to April.

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President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.

Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.

Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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CLINICAL PROBLEM OF ALLERGY IN RELATION TO CONJUNCTIVITIS AND IRITIS

ALAN C. WOODS, M.D.

BALTIMORE

The importance of allergy, or an altered state of reactivity to specific agents, has been stressed repeatedly in the recent ophthalmic literature. Much of this literature deals with purely experimental lesions of the eye, and a small amount deals with clinical investigation. Rather broad deductions appear to have been made from these experimental and clinical findings, and at present clinical ophthalmologists are constantly confronted with the questions of therapeutic desensitization and various forms of vaccine treatment for patients with inflammatory lesions of the conjunctiva and anterior part of the uveal tract.

I have no wish to minimize the importance of allergic reactions in these conditions. However, the questions involved are complicated and in many instances not clearly understood, and often lie more in the domain of the immunologist and of the bacteriologist than in the province of the ophthalmologist. It is my purpose in this paper to outline, from the point of view of the ophthalmologist, the present state of knowledge on the relation of allergy to conjunctivitis and iritis and to indicate the clinical application of this knowledge.

ALLERGY AND CONJUNCTIVITIS

A large number of authors—Koutseff,¹ LaGrange and Delthil,² Krückmann,³ Berneaud,⁴ Blake,⁵ Dejean and Temple,⁶ and numerous others—have reported cases of allergic conjunctivitis. Koutseff divided

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

Read before the meeting of the American Academy of Otolaryngology and Ophthalmology, New York, Sept. 29, 1936.

1. Koutseff, A.: Contribution à l'étude des conjonctivites allergiques non infectieuses, *Zentralbl. f. d. ges. Ophth.* **33**:333, 1935; Les conjonctivites allergiques non infectieuses envisagées dans le cadre des allergies en général, *Bull. Soc. d'ophth. de Paris*, April 1935, p. 267.

2. LaGrange, H., and Delthil, S.: Les réactions allergiques de la conjonctive oculaire, *Ann. d'ocul.* **170**:1009 (Dec.) 1933.

3. Krückmann, E.: Ueber Allergie in der Augenheilkunde, *Klin. Monatsbl. f. Augenh.* **92**:809, 1934.

(Footnotes continued on next page)

allergie conjunctivitis into two distinct clinical types, both due to direct contact of the sensitized conjunctiva with the specific allergen. The first is the sudden, edematous type, characterized by a sudden onset, edema, conjunctival congestion and lacrimation. Koutseff stated the belief that this is due to allergens suspended in the atmosphere, notably the pollens. The second is the follicular, eezematous type, which comes on slowly, characterized by eezema of the lids and neighboring skin, and is produced most frequently by drugs such as butyn, atropine and the various collyria and salves. LaGrange and Delthil² described three types: first, a transitory form, characterized by edema of the palpebral and bulbar conjunctiva, which is sudden in both its appearance and its disappearance; second, chronic blepharoeconjunctivitis, with a tendency to eczema of the lids, and, third, a chronic follicular type. LaGrange and Delthil² stated the belief that the endocrine glands and the vegetative nervous system are concerned in cases of allergic conjunctival disorders. Krückmann³ likewise expressed the opinion that there are three types of allergic conjunctivitis. The first is the sudden, edematous type, more or less strictly localized, associated with glassy edema of the conjunctival fold, without redness of the margins of the lid or evidence of furunculosis or hordeolum, which is sudden in both its appearance and its disappearance; the second is blepharoeconjunctivitis associated with eezema of the lids, and the third is simple hyperemia that lasts for weeks, without much secretion of mucus. Krückmann expressed the belief that vernal conjunctivitis occupies a special position. He also made the point that the conjunctivitis that appears in hypersensitive persons after the ingestion of certain foodstuffs, notably shell-fish and lentils, is indistinguishable from the conjunctivitis produced at times by atropine. This classification of three types of allergic conjunctivitis is excellent, but the clinical appearance gives only a slight hint as to the nature of the causative allergen, which may be pollen, animal dust, food, or bacterial toxins or their derivatives.

The best known example of the sudden edematous type of allergic conjunctivitis is that seen in association with asthma and hay fever. The mucous membrane of the eye, the conjunctiva, participates in the general hypersensitivity of the mucous membranes of the upper respiratory passages and, like them, reacts with sudden inflammatory edema when in contact with the specific sensitizing substance, the air-suspended allergens. There are usually chemosis of the palpebral and bulbar conjunctiva, congestion, profuse lacrimation and a nonpurulent secretion without bacteria. The attack subsides quickly after the instillation of epinephrine hydrochloride.

4. Berneaud, G.: *Allergische Augenerkrankungen*, Ztschr. f. Augenh. **78**:193, 1932.

5. Blake, E. M.: *Allergic Reaction of the Eyelids*, Arch. Ophth. **53**:272, 1924.

6. Dejean, C., and Temple, J.: *Some Special Cases of Follicular Conjunctivitis*, Bull. et mém. Soc. franç. d'opht. **46**:143, 1933.

The classic example of the edematous, eczematous type of allergic conjunctivitis is that caused by drugs. This presents little diagnostic difficulty. The eczematous cutaneous reaction and edema of the lids, the profuse lachrimation and the conjunctival congestion, usually associated with a slight degree of chemosis, present a characteristic picture. The offending drug can usually be identified without difficulty, for patients use few drugs in their eyes. This type of conjunctivitis is always accompanied by marked generalized hypersensitivity of the skin, which can be demonstrated by the ordinary patch test—the application of a salve containing the drug to the skin of the leg, this area then being covered with a bandage for twenty-four hours. If the patient is hypersensitive to the drug, marked dermatitis will occur at the site of application. By this test a suspected sensitivity can be determined before a drug is used. An occasional patient is sensitive to the oily base usually employed in salves. This type of conjunctivitis subsides slowly, in several days, after the withdrawal of the offending drug. A drug that is entirely innocuous on first administration may gradually acquire the properties of a sensitizing allergen. Landsteiner⁷ showed that simple inert substances, even metals, may combine with native protein to form sensitizing substances. This is probably the explanation for the development of hypersensitivity to drugs by the conjunctiva. The offending drug, in contact with the lacrimal secretion, forms a foreign compound capable of producing the specific sensitization.

The third type of allergic conjunctivitis is chronic, recurrent, irritative conjunctivitis, often associated with low grade folliculosis, with rather sharp exacerbations and with consistently normal bacteriologic findings. The responsible allergens may be pollens, animal dust, foods or bacterial allergens. The detection and demonstration of the responsible allergens present a difficult diagnostic problem. Attention is drawn to such an etiologic possibility by the clinical picture of the condition, the normal character of the bacterial flora, the resistance of the conjunctivitis to ordinary treatment, such as astringents and collyria, and the correction of refractive errors and muscular anomalies. In this type of conjunctivitis the conjunctival reaction is often the only clinical evidence of tissue allergy.

The first step in the detection of the responsible allergen or allergens is a careful taking of the history. Are there other allergic manifestations; is there a history of allergy in the patient or the family, and was there any constant common factor in the patient's life and diet previous to the exacerbation? The second step is to test the patient against the specific individual and group allergens.

The best test for specific sensitivity is the intracutaneous injection of specific allergens. This is less misleading in its results and permits

7. Landsteiner, K.: Experiments on Anaphylaxis to Azoproteins, *J. Exper. Med.* **39**:631, 1924.

greater accuracy than does the scratch method. The usual diagnostic dose of pollens, foods and animal dusts is 0.03 cc. The diagnostic intracutaneous dose of bacterial toxins is 0.1 cc. of different dilutions. The reactions are read in fifteen minutes. Several tests can be made at one time. There are two distinct major groups of allergens against which the patient must be tested: first, the group comprising the pollens, foods and animal dusts, and a few specific allergens, and, second, the bacterial toxins.

Specific Allergens.—The first general group of allergens, the pollens, foods and animal dusts, may be divided into twelve subgroups: the common pollens, the tree pollens, the rare pollens, epidermals and inhalants, vegetables, fruits, grains and spices, dairy products, meats, sea food and nuts, and special allergens, such as intestinal parasites, silk and rayon. Each of these subgroups contains from five to twenty-two allergens, in all, but one hundred and forty different substances against which the patient must be individually tested, often with different dilutions. In addition to these twelve groups, there are two other groups of composite antigens, made up of botanically related foods. In these two groups there are eighteen composite antigens, some containing as high as eight allergens. If a patient is sensitive to one of these group allergens he must be tested against each of the individual components of the group.

It is therefore apparent, when one considers the great number of allergens necessary to determine a suspected hypersensitivity and the complexity of the task involved, that such a study is beyond the province of the clinical ophthalmologist. The patient must be referred to an immunologist for the determination of the allergic status as regards the pollens, foods, animal dusts and inhalants. When such a study has been completed, the ophthalmologist is faced with the problem that the patient often shows a multiple sensitivity; that is, he is sensitive to a number of widely different allergens. There is also some evidence that cutaneous sensitivity and conjunctival sensitivity may not be parallel. Frequently, therefore, it must be determined to which allergen, if any, there is an especial conjunctival sensitivity. This is determined by the ophthalmic test, the instillation into the conjunctival sac of a 1:10 or 1:100 dilution of the various allergens to which cutaneous sensitivity has been demonstrated. The eye should be quiet and the corneal epithelium intact, lest allergic keratitis be produced. A positive ophthalmic test is evidenced by prompt conjunctival congestion and lacrimation. If such a test is positive, the eye should be immediately irrigated and a drop of solution of epinephrine hydrochloride instilled.

After a diagnosis of allergic conjunctivitis due to sensitivity to pollens, foods or animal dusts, there arises the question of treatment. Theoretically, desensitization by repeated subcutaneous injections of the

specific allergen is indicated. However, the length of time necessary to achieve such desensitization, the tendency of the hypersensitivity and symptomatology to recur and the uncertainty of obtaining complete conjunctival desensitization are discouraging factors. The knowledge of ocular therapy is not yet advanced sufficiently to warrant the attempt at local desensitization by such methods as repeated local administration of the specific allergens. To my mind, the most important step is the education of the patient to avoid the specific intoxicating allergen. Thus, a patient with a cutaneous and conjunctival hypersensitivity to feathers must be taught to keep rigorously away from all poultry and birds and to sleep on a kapok pillow, while a patient with food allergy must be cautioned to abstain from the specific food. The change in the clinical picture of the eyes after such simple advice is followed is often astounding. The only local treatment indicated is cleansing the eyes with any bland collyrium or salt solution and the use of weak astringents with a little epinephrine added. Until it is demonstrated that such expectant treatment is unpractical or without avail I do not feel justified in undertaking specific desensitization.

Bacterial Toxins.—Staphylococcus Toxin: The second large group of allergens are the bacterial toxins and nucleoproteins. The most important of these, from the standpoint of the ophthalmologist, is staphylococcus toxin. With a small amount of training an abnormal hypersensitivity to staphylococcus toxin can be determined by the ophthalmologist himself. Studies with other toxins and studies with bacterial proteins lie in the domain of the bacteriologist.

While staphylococcus toxin is lethal in small doses, it is not in itself irritative to a normal skin or normal mucous membrane. It produces irritative symptoms only in hypersensitive tissues. The probable mechanism of conjunctivitis due to staphylococcus toxin may be conceived of as follows: infection with a toxin-producing staphylococcus is followed by local absorption of the toxin and resultant tissue hypersensitivity. Fluctuation in the growth of the infecting bacteria is accompanied by changes in the toxin formation. The absorption of toxin by the hypersensitive conjunctiva results in an allergic inflammatory reaction, which in its very nature is a chronic process, with periods of remission and exacerbation corresponding to the fluctuations in the formation of toxin and the exhaustion and recurrence of the local hypersensitivity. Even if the infecting organism is eradicated from the conjunctival sac the tissue hypersensitivity to toxin remains, and fresh infection with the same organism, which must be a common occurrence, is followed by a recurrence of the conjunctivitis. Hypersensitivity to the specific protein of the bacterial bodies of staphylococci is probably unimportant in the etiology of allergic conjunctivitis.

Burky⁸ has studied extensively the staphylococcic infections of the conjunctiva. He found that the staphylococci isolated from the normal and pathologic conjunctiva are of three different types. The first is an ordinary nonpathogenic albus type that occurs in the normal eye and that produces no symptoms. The second is a pus-producing pigmented organism found in cases of purulent conjunctivitis, furuncles and pustular blepharitis. The third organism, termed by Burky "Ha," is a hemolytic toxin-producing staphylococcus. This organism produces a strongly potent toxin, 0.1 cc. per kilogram of body weight given intravenously being the lethal dose for an adult rabbit, with death occurring in twenty-four hours. Studies on man and animals by Burky and others have shown that the skin of young persons is not sensitive to this toxin. As human beings and animals grow older, however, and are exposed to constant and repeated infection with staphylococci, a cutaneous sensitivity of greater or lesser degree to this toxin may gradually develop. Likewise, substances develop in the blood which give a positive complement-fixation reaction against an antigen made from this organism. Studies on man and animals have indicated that a positive complement-fixation reaction and a low degree of cutaneous sensitivity to the toxin indicate a comparatively high degree of immunity to the toxin and to staphylococcic infections, while a negative or weak complement-fixation reaction and a high degree of cutaneous sensitivity indicate marked susceptibility to such infections. A person or an experimental animal may be actively protected against the action of this toxin by being given repeated intracutaneous injections of the toxin, desensitization and immunization thus being accomplished, or the subject may be passively protected by the administration of a prepared serum containing the specific staphylococcus antitoxin. Fundamentally important is the fact that the protection resulting from such active or passive immunization is potent not only against the specific toxin but against all staphylococcic infections.

Clinically, there is a large group of patients with recurrent, chronic conjunctivitis and marginal blepharitis in whom this susceptibility to staphylococcus toxin is apparently the specific causative factor. Practically, how may such a condition be detected and treated?

A presumptive diagnosis of allergic conjunctivitis due to hypersensitivity to staphylococcus toxin is made on the criteria of chronic, recurrent nonpurulent conjunctivitis, often associated with the following: folliculosis; marginal blepharitis; reddened, swollen margins of the lids; frequently, slight secondary corneal changes; the elimination of other obvious causes for the conjunctivitis; the isolation of a toxin-

8. Burky, E. L.: Staphylococcus Toxin and Antitoxin, *Internat. Clin.* **3**:258 (Sept.) 1936.

producing staphylococcus from the conjunctiva; the presence of a high degree of cutaneous sensitivity to staphylococcus toxin, and, usually, a low degree of complement fixation against the staphylococcus antigen. Conjunctival cultures taken after the omission of the use of antiseptics or during exacerbations of the inflammation usually show the toxin-producing organism, although sometimes even when all the other criteria for diagnosis are present the cultures show no staphylococcus on repeated tests, the organism itself presumably having been eradicated but leaving the local hypersensitivity.

The cutaneous sensitivity is determined, first, by the intracutaneous injection of 0.1 cc. of a 1:100 dilution of the toxin and, later, by the injection of a 1:1,000 or a 1:10 dilution, depending on whether the first injection has given a positive or a negative result. A normal person reacts to the 1:100 dilution with an area of erythema approximately from 3 to 4 cm. in diameter. Reactions greater than this or reactions to a higher dilution are regarded as indicating undue hypersensitivity. A strongly positive reaction is sometimes accompanied by a little necrosis at the site of the injection. The complement fixation test is carried out by the usual technic, a 1:8 dilution of the staphylococcus toxin and a 1:8 dilution of the filtrate of broth cultures of a pus-producing staphylococcus being used as antigens and a 1:8 dilution of the filtrate of the nonpathogenic organism being used as a control.

The indicated treatment of such a conjunctivitis is active immunization or desensitization with the staphylococcus toxin. According to the degree of cutaneous sensitivity present, a dilution of the toxin is selected. This is usually 1:100, but for the extremely hypersensitive person with a reaction of more than 8 by 8 cm. a 1:1,000 or a 1:10,000 dilution is used. All the injections are given intracutaneously. The initial dose is 0.1 cc. This dose is given every four days until the local reaction becomes minimal. Usually from four to ten injections are needed for this point to be reached. When a minimal local reaction to the first dilution is reached, the dilution of the toxin is then concentrated ten times, the concentration being increased from 1:1,000 to 1:100 or from 1:100 to 1:10, and 0.1 cc. of this stronger concentration is given until again a minimal reaction is obtained. This is continued until the patient can tolerate 0.1 cc. of pure toxin with a minimal reaction. This is apparently the point of desensitization. During such treatment occasional variations are sometimes seen: A patient may react violently after one injection, while after subsequent injections there is only a minimal reaction. Focal inflammatory conjunctival reactions may come on a few hours after the injection of the toxin and last from twenty-four to thirty-six hours.

In the last few years in the Wilmer Ophthalmological Institute over fifty patients with allergic conjunctivitis due to staphylococcus toxin

have been treated. The results are difficult to tabulate on account of the varying degrees of sensitivity and ocular involvement and the difficulty of achieving and maintaining desensitization. In general the statement can be made that the therapeutic results have been surprisingly good and that this form of treatment has offered in otherwise hopeless and intractable cases a real therapeutic weapon that often gives spectacularly brilliant results.

In several instances passive immunization by means of the injection of an antitoxin serum intramuscularly has been used for patients with staphylococcic septicemia, and in one case of a person with blind cataract who had allergic blepharoconjunctivitis resulting from staphylococcus toxin. In this case one eye had been operated on elsewhere and lost because of postoperative infection. Local treatment to eradicate the staphylococcus from the conjunctiva was totally unavailing. The patient was finally given 20 cc. of a strongly antitoxic rabbit serum intramuscularly and was operated on twelve hours later. He received further daily injections of the antitoxin for five days until signs of a general reaction occurred, after which the treatment was discontinued. Recovery from the operation was uneventful; there was no reaction or infection, although the conjunctival sac constantly contained a virulent toxin-producing staphylococcus.

There is one caution to be emphasized in the use of any foreign antiserum in the treatment of patients with disease of the external part of the eye. A severe reaction may be accompanied by marked edema of the lids. If there is corneal involvement or if such involvement is feared, the sealing of the eye by edema of the lids may be hazardous. For this reason passive immunization is not recommended unless the circumstances are such that there is no other course open.

B, Streptococcus Toxin: There is little actual information on the importance of the toxins of streptococci or other organisms in the production of allergic conjunctivitis. Streptococci produce certain known toxins, hemolysins, leukocytin and scarlatina and erysipelas toxins. Actual allergy to such toxins is possible, and it is conceivable that allergic inflammations of the external part of the eye might result from such sensitization. Scholtz⁹ has reported one case of conjunctivitis in which he believed that the condition was clearly due to allergy to streptococcus toxin. However, the whole question of hypersensitivity to streptococci and their toxins is so complicated that it is impossible to speak with any degree of certainty on the subject.

Primarily, there are two types of sensitivity to streptococci and their products. The first is the sensitivity shown following the intra-

9. Scholtz, H. G.: *Schwerste stomakake Conjunctivitis, Rhinitis und Bronchitis bei allergischer Diathese*, München. med. Wchnschr. **79**:916 (June 3) 1932.

cutaneous injection of suspensions of the actual killed organisms. Such reactions when positive are of the type of delayed tuberculin reactions, coming on after twenty-four hours, and are probably indicative of actual sensitivity to the nucleoprotein of the organisms. The second is the immediate cutaneous sensitivity shown after the intracutaneous injection of filtrates of these organisms, which may indicate sensitivity to specific toxins, to the specific carbohydrate, to the products of bacterial growth or even to substances in the culture mediums. A large percentage of entirely normal persons show positive cutaneous reactions both to suspensions of the bacterial bodies and to the filtrates. Thus, MacKenzie and Hanger¹⁰ expressed the belief that cutaneous hypersensitiveness to such bacterial substances was in general a function of age or an index of normal exposure to infection rather than an index of susceptibility to any specific substance or disease. Numerous other investigators have studied the problem, and there is some evidence that the hypersensitivity is greater and more frequent in certain diseases, notably, rheumatoid arthritis, which is supposedly related to focal infection.

The diagnostic problem is further complicated by the multiplicity of strains of hemolytic streptococci, each of which must be studied as an individual entity. Of the various groups of hemolytic streptococci pathogenic for man, group A contains over thirty strains, group B contains three strains and group F contains four strains. Swift, Lancefield and Goodner¹¹ stated the belief that different specific strains do not cause different disease conditions but that the reaction to infection with any strain may vary from generalized infections to simple pharyngitis, according to the individual reaction of the infected patient. The magnitude of the task of attempting to detect any specific sensitivity responsible for ocular inflammation can be realized when one considers the high percentage of nonspecific reactions and the large number of strains against which the subject is to be tested.

In actual practice the detection of a streptococcus in the bacterial flora of a patient with chronic conjunctivitis might bring up the question of whether hypersensitivity to the bacterial protein or the products of bacterial growth is responsible for the local inflammation. Since there is no streptococcus toxin or vaccine which gives blanket protection, the only practical method of attacking the problem is the preparation of a killed suspension and of filtrates of cultures of the organism isolated from the patient and testing the patient and controls against such antigens. If the patient shows a reaction to either the killed organism

10. MacKenzie, G. M., and Hanger, F. M., Jr.: Allergic Reactions to Streptococcus Antigens, *J. Immunol.* **13**:41, 1927.

11. Swift, H. F.; Lancefield, R. C., and Goodner, K.: The Serologic Classification of Hemolytic Streptococci in Relation to Epidemiologic Problems, *Am. J. M. Sc.* **190**:445, 1935.

or to the filtrates greater than that observed in the controls, the interpretation may be that hypersensitivity to this organism may be concerned in the clinical picture.

As nothing is known of any protective effect of the injection of a streptococcus toxin, the only treatment of supposed allergic conjunctivitis caused by hypersensitivity to a specific streptococcus is vaccine therapy with the killed organisms. Swift¹² and his co-workers have shown that, while intracutaneous injections of killed streptococci tend to sensitize rabbits, intravenous injection of the organism produces an immunity. In the treatment of hypersensitive patients with rheumatoid arthritis Wainwright¹³ employed with success the intravenous injection of a salt solution suspension of the heat-killed organisms. An initial dose of 0.5 cc. containing approximately 5,000,000 heat-killed organisms was given. The following doses were determined by the amount of reaction to the first dose. In general, the injections were given at four day intervals, the dose being increased 0.5 cc. at each injection and kept below that by which general symptoms were produced. The patients were treated for periods of from two months to one year. If one must use a streptococcus vaccine, this appears to be the logical procedure.

ALLERGY AND VERNAL CONJUNCTIVITIS

Vernal conjunctivitis is generally believed to be an allergic disturbance. The reasons for this belief are the seasonal occurrence of the attacks, the comparative absence of bacteria in the secretions, the tendency of the disease to attack the young, the specific hypersensitivity and general allergic tendency of the patients and the presence of eosinophils in the conjunctival secretion. While eosinophilia is not pathognomonic of an underlying allergy, it is either dependent on or closely related to the absorption of protein and is usually found in allergic subjects.

The assumption that an underlying allergy is responsible for vernal catarrh is strengthened by the experimental demonstration that the conjunctiva is susceptible of specific sensitization and by the histologic study of the follicles on the bulbar and palpebral conjunctiva. The follicle which appears on the eye near the limbus has the general histologic picture of a phlyctenule, which has rather definitely been proved to be an allergic manifestation. The second type of follicle, which appears on the palpebral conjunctiva, consists of papillary swelling

12. Swift, H. F.: The Pathogenesis of Rheumatic Fever, *J. Exper. Med.* **39**:497, 1924.

13. Wainwright, C. W.: Treatment of Chronic Rheumatoid Arthritis with Streptococcus Vaccine on the Basis of Skin Sensitivity, *J. A. M. A.* **103**:1357 (Nov. 3) 1934.

associated with proliferation of connective tissue, with infiltration by lymphocytes, plasma cells and eosinophils. This reaction is somewhat comparable to that seen in the bronchial walls in cases of asthma and in the skin in cases of eczema and has been spoken of as an allergic reaction characterized by persistent granulation.

In 1933 LaGrange and Delthil¹⁴ published a short monograph on conjunctivitis and vernal catarrh and in the last three years have published other papers¹⁵ on the same subject. These authors stated that they definitely regard vernal conjunctivitis as an allergic disease of the pseudofollicular type of conjunctival allergy. Their conclusion is based principally on clinical observation and study. They accept the specific sensitization of the conjunctiva as a fact proved by experiments. The classic allergic syndrome of vernal conjunctivitis is: (1) the clinical picture, the recurrences and the associated itching; (2) the climatic and seasonal incidence; (3) the frequent association of vernal conjunctivitis with other allergic conditions, such as asthma, hay fever, urticaria and angioneurotic edema; (4) the absence of bacteria and inclusion bodies and the presence of eosinophils in the conjunctival secretions; (5) the cutaneous sensitivity and the positive Prausnitz-Küstner reaction to the specific allergen, shown by the patient; (6) the reaction of the affected conjunctiva, first, to epinephrine and, second, to the specific allergen; (7) the frequent association of endocrine disturbances and an imbalance of the vagosympathetic system.

LaGrange and Delthil¹⁴ stated that they are firmly of the opinion that other factors influence the allergic diathesis of the affected persons. These factors are heredity and endocrine disturbances. These authors believe that disorders of the endocrine glands induce a secondary imbalance of the vagosympathetic system and that this predisposes to sensitization of the tissues and the resultant allergic reactions. They believe that the conjunctival localization of the sensitivity and allergic reaction in vernal conjunctivitis is only another manifestation of the elective localization of cutaneous allergy and that possibly the direct exposure of the conjunctiva to direct sensitization may be responsible for this.

In support of these views these authors report in detail a large number of clinical cases of allergic and vernal conjunctivitis illustrating the various points emphasized in their allergic syndrome, especially endocrine disorders, affecting principally the gonads, alterations in the oculocardiac reflex, and certain rather startling results obtained by organotherapy with various endocrine preparations. Especially interest-

14. LaGrange, H., and Delthil, S.: *Les conjonctivites de nature anaphylactique*, Paris, Gaston Doin & Cie, 1932.

15. LaGrange and Delthil.² LaGrange, H.: *Le rôle de l'allergie dans certaines conjonctivites*, Bull. Soc. d'opht. de Paris, April 1935, p. 230.

ing is LaGrange's last observation in 1935, which indicates that patients with vernal catarrh show evidences of the vascular shock reaction termed by Widal the *phénomènes colloïdoclasiques*, which is seen constantly in cases of experimental anaphylactic shock.

This is the evidence so far amassed that vernal conjunctivitis is an allergic disease—the clinical picture, the absence of bacteria, the seasonal occurrence, the experimental demonstration that the conjunctiva is susceptible of specific sensitization, the evidence that patients with the disease have a much higher percentage of associated allergy than do normal patients, the demonstration of a pathologic hypersensitivity to allergens in the greater percentage of such patients and the pathologic picture of the diseased conjunctiva.

From the point of view of the clinician, how must such an assumption influence one's approach to the study and treatment of this disease? The problem is little different from that already outlined for other forms of allergic conjunctivitis. The essential thing is to find the specific allergen or allergens to which the patient is hypersensitive and endeavor, first, to isolate the patient from these substances and, second, since vernal conjunctivitis is a recurrent, chronic disease, to achieve and maintain desensitization. Such procedures are no contraindication to other routine therapy, the use of astringent collyria, treatment with radium or even organotherapy. Personally, I believe that the present knowledge of endocrinopathies and endocrine therapy has not yet progressed to the point where organotherapy is an indicated procedure except when used in a very limited degree.

ALLERGY AND FOCAL INFECTION

Recent experimental work has indicated that certain types of recurrent uveitis and iritis may be allergic reactions dependent on sensitization and intoxication of the uveal tissue by bacterial derivatives. In 1933 I¹⁶ summed up the evidence in favor of this proposition. This idea was primarily suggested by the frequency with which sterile cultures were obtained from the iris and aqueous in cases of acute iritis and by the flare-ups in the ocular inflammation which frequently followed the subcutaneous administration of bacterial vaccines. Experimentally this hypothesis found strong confirmation in the work of Swift and Derick,¹⁷ Schultz and Swift,¹⁸ Julianelle,¹⁹ Seegal and

16. Woods, Alan C.: *Allergy and Immunity in Ophthalmology*, Baltimore, Johns Hopkins Press, 1933.

17. Swift, H. F., and Derick, C. L.: Hyperergic Tissue Response to Non-Hemolytic Streptococci, *Proc. Soc. Exper. Biol. & Med.* **25**:222, 1927-1928.

18. Schultz, M. P., and Swift, H. F.: Reaction of Rabbits to Streptococci: Comparative Sensitizing Effect of Intracutaneous and Intravenous Inocula in Minute Doses, *J. Exper. Med.* **55**:591, 1932.

Seegal²⁰ and others. These investigators had shown that repeated intracutaneous injections of living streptococci or the absorption of bacterial products from an agar focus implant produced in the experimental animal a definite hypersensitivity of the ocular tissues, and later contact of the sensitized eye with the specific bacteria or bacterial products resulted in ocular inflammation. This was termed the ophthalmic reaction. Since 1933 the experimental foundation for this theory has been still further strengthened. Seegal and Seegal and Khorazo²¹ showed that if eyes sensitized by the direct injection of a specific protein in the anterior chamber were allowed to rest until all traces of specific protein had disappeared from the anterior chamber and were then activated by an intravenous injection, traces of the specific antigen again appeared in the anterior chamber. This finding gave an explanation for the resensitization of eyes after the primary reaction and readily explains why recurrent attacks of inflammation may take place. Seegal and Seegal²² also showed that if the eyes of experimental animals were subjected to some nonspecific inflammation or insult at the time a foreign protein was circulating in the blood stream, the circulating foreign protein was absorbed directly into the eye, and sensitization of the eye resulted. Julianelle, Morris and Harrison²³ found certain cases of vascularization of the cornea in which the condition was an allergic phenomenon. Eyes sensitized by repeated intracutaneous and subconjunctival injections and later traumatized and exposed to the absorption of the specific antigens showed corneal vascularization. This these authors obtained with certain bacterial antigens, with egg albumin and with the purified nucleoprotein of various organisms. Such vascularization was likewise obtained by the repeated

19. Julianelle, L. A.: Reactions of Rabbits to Intracutaneous Injections of Pneumococci and Their Products: V. The Development of Eye Reactivity to Derivatives of Pneumococci, *J. Exper. Med.* **51**:633, 1930; VI. Hypersensitiveness to Pneumococci and Their Products, *ibid.* **51**:643, 1930.

20. Seegal, D., and Seegal, B. C.: Local Organ Hypersensitiveness: I. Experimental Production in the Rabbit Eye, *Proc. Soc. Exper. Biol. & Med.* **27**:390, 1930.

21. Seegal, B. C.; Seegal, D., and Khorazo, D.: Local Organ Hypersensitiveness: V. The Fate of Antigen and the Appearance of Antibodies During the Development of Hypersensitiveness in the Rabbit Eye, *J. Immunol.* **25**:207, 1933.

22. Seegal, B. C., and Seegal, D.: Local Organ Hypersensitiveness: VI. An Indirect Method for Its Production in the Rabbit Eye, *J. Immunol.* **25**:221, 1933.

23. Julianelle, L. A.; Morris, M. C., and Harrison, R. W.: Studies on Vascularization of the Cornea: I. Sensitization of Cornea of Rabbits to Bacteria, *J. Immunol.* **26**:267 (April) 1934; II. Sensitization of Cornea of Rabbits to Proteins, *ibid.* **26**:281 (April) 1934. Julianelle, L. A.; Harrison, R. W., and Morris, M. C.: III. Sensitization of Cornea of Monkeys, *ibid.* **26**:295 (April) 1934. Julianelle, L. A.; Morris, M. C., and Harrison, R. W.: IV. Question of Passive Corneal Hypersensitiveness, *ibid.* **26**:303 (April) 1934.

application of living cultures to the lightly scarified cornea, becoming apparent after the ninth exposure. This allergic reaction appeared as a local manifestation and had no relation to the general antibody content of the blood serum or to the heightened reactivity of the skin to the specific allergens. In 1934 Brown²⁴ repeated the work of Swift and Derick and their co-workers. His experiments indicated that the ocular sensitivity resulting from infected agar implants might occur as early as the first to the third day. Further, eyes directly sensitized by killed streptococci could be activated by remote infected agar implants, indicating that bacterial products diffusing out from a chronic focus of infection could produce inflammatory intra-ocular reactions in sensitized intra-ocular tissues. A further step forward was made by experiments of MacLean²⁵ in the Wilmer Ophthalmological Institute. MacLean confirmed the results of the earlier work of Swift and Derick and their co-workers on the ophthalmic reaction and succeeded in producing a transient inflammatory uveal reaction in the untraumatized eye through intracutaneous sensitization and intravenous intoxication with living streptococci. Brown²⁶ recently showed that multiple sensitization of eyes could be obtained experimentally by the injection of different allergens, both bacterial and protein. He further showed that the injection of an excessive dose of one of these allergens resulted in the subsequent inhibition of intoxication by other allergens. If typhoid vaccine was used as an "interfering allergen" and the antibody content of the aqueous was artificially increased, the intoxicating effects of other allergens was inhibited. This offers a possible new explanation for the effect of nonspecific protein therapy—that the mobilization of nonspecific antibodies in the aqueous inhibits the intoxicating effects of other allergens to which the eye may be sensitive.

A number of observers, notably, Julianelle²⁷ and Seegal, Heidelberg and Jost²⁸ have endeavored to determine to which portion of the bacterial antigen mosaic the ocular sensitivity was due. Julianelle showed that the sensitivity resulting from the intracutaneous injections was apparently due to the nucleoprotein of the bacterial body, and Seegal and Heidelberg, working on the formation of precipitins with

24. Brown, A. L.: Chronic Uveitis: Bacteriologic and Immunologic Considerations, *Arch. Ophth.* **12**:730 (Nov.) 1934.

25. MacLean, A. L.: Experimental Iritis: The Ocular Reactions in Rabbits Sensitized to *Streptococcus Viridans*, *Tr. Am. Ophth. Soc.*, 1936, to be published.

26. Brown, A. L.: *Tr. Am. Ophth. Soc.* **33**:435, 1935.

27. Julianelle, L. A.: Reactions of Rabbits to Intracutaneous Injections of Pneumococci and Their Products, *J. Exper. Med.* **51**:441, 1930.

28. Seegal, D.; Heidelberg, M., and Jost, E. L.: The Formation of Precipitin for the Group A Specific Carbohydrate of *Streptococcus Hemolyticus* in Rabbits Injected Intravenously and Subcutaneously. *J. Immunol.* **27**:211. 1934.

the specific carbohydrate of *Streptococcus haemolyticus*, confirmed the results of Julianelle's work, their experiments indicating that the sensitivity was chiefly due to the nucleoprotein fraction and not to the specific carbohydrate. In 1929 Tillet and Francis,²⁹ working with patients with lobar pneumonia, showed that the immediate edematous reactions of the skin to pneumococci were due to the specific carbohydrate, while the delayed tuberculin type of reaction was due to the nucleoprotein of the pneumococcus.

Berens and his co-workers³⁰ studied from the clinical point of view the cutaneous reactions and the agglutinin titer of patients with ocular diseases against various organisms isolated from the nose, infected teeth and tonsils of these patients. They expressed the belief that such sero-bacteriologic studies give information as to the identity of the organism responsible for the ocular lesion and that such reactions are of decided value to the ophthalmologist studying diseases of the eye due to focal infection and likewise might indicate the specific bacteria from which vaccines for therapeutic use should be prepared. Benedict³¹ has called attention to the relationship between infection in the female pelvis and certain recurrent ocular diseases and to the beneficial effects obtained with autogenous vaccines. There are other reports, too numerous to mention, on the use of autogenous vaccines isolated from foci of infection in the treatment of recurrent ocular disease.

Thus it is evident that a rather substantial experimental background has been built up for the hypothesis that recurrent iritis or recurrent uveitis may be an actual allergic manifestation due to sensitization and intoxication from bacterial allergens. Confronted with this experimental evidence that allergy may be concerned in chronic, recurrent uveitis and with the clinical evidence of heightened cutaneous reactivity to certain bacteria harbored in suspected foci of infection, what should be the position of the clinical ophthalmologist on the problem? Ophthalmologists are constantly confronted with the suggestion that the skin of the patient be tested against all organisms isolated from any focus of infection or even from the gastro-intestinal tract and that the organisms to which it may react be assembled into an autogenous vaccine for therapeutic use. Such vaccine therapy, based solely on cutaneous reactivity to bacteria, in the present state of knowledge, is to my mind unjustifiable and scientifically indefensible. In the normal course of

29. Tillet, W. S., and Francis, T.: Cutaneous Reactions to the Polysaccharides and Proteins of *Pneumococcus*, *J. Exper. Med.* **50**:687, 1929.

30. Berens, C.; Connolly, P. T., and Chapman, G. H.: Focal Infection in Diseases of the Eye: I. Report of Certain Laboratory Examinations, *Am. J. Ophth.* **17**:1094, 1934.

31. Benedict, W. L.: The Relation of Infections of the Pelvic Organs and Diseases of the Eye, *Am. J. Ophth.* **16**:665, 1933.

life any average human being is bound to become hypersensitive and show a cutaneous reactivity to a great host of common bacteria to which he is constantly exposed. All studies such as those of Derick and Fulton,³² MacKenzie and Hanger,¹⁰ Birkhaug³³ and numerous other investigators have shown that the greater percentage not only of diseased persons but of normal persons have a cutaneous sensitivity to common organisms, such as streptococci and their derivatives. If one accepts such cutaneous reactivity as a criterion for vaccine therapy, there is no disease in the whole gamut of pathologic conditions of man which should not be so treated. The patient has no chance of escape. Further, it is not clearly known whether the vaccine should be prepared from the bacterial bodies or from the filtrates. In the case of staphylococci, as has already been pointed out, all the evidence indicates that the maximum protective effect is obtained by a properly prepared filtrate of toxin, while in the case of streptococci the present indications are for the use of a vaccine prepared from the bacterial bodies. To determine the proper preparation of vaccine each suspected organism must be studied separately; otherwise one is guilty of the rankest kind of shotgun therapy. In the present state of knowledge there appears to be little justification for the use of a vaccine prepared from the intestinal flora. In the case of organisms isolated from a focus of infection, the only indication for the use of a vaccine would be, first, that there is some reason because of the nature of the illness and the exclusion of other causal factors to suspect one individual organism and, second, that the affected person shows a far more intense cutaneous reactivity to this organism than would normally be expected or than is shown by normal controls. Only with these criteria, I believe, is the use of a vaccine justified in cases of recurrent uveitis, and then certainly not until the first duty—the eradication or treatment of the primary focus of infection—has been accomplished. In short, while there is much experimental evidence to indicate that certain cases of recurrent uveitis may be due to bacterial allergy from a focus of infection, such a proposition is far from being proved, and with the present limited knowledge there is little justification for the current promiscuous use of vaccine therapy in cases of recurrent uveitis.

CONCLUSIONS

Allergic conjunctivitis may appear as: (1) sudden transitory conjunctivitis, characterized by edema, chemosis and conjunctival congestion and injection; (2) chronic blepharoconjunctivitis with a tendency

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to eczema of the lids, or (3) chronic, recurrent conjunctivitis, often complicated by folliculosis and blepharitis.

Such conjunctivitides may be due to pollens, food, animal dusts, inhalants or bacterial toxins and proteins. The diagnosis is made on the basis of demonstration of a specific cutaneous hypersensitivity and may be confirmed by an ophthalmic test. Treatment consists of isolation of the patient from the offending allergen and specific desensitization.

The present evidence indicates that vernal catarrh is an allergic disturbance.

While there is much experimental evidence indicating that recurrent iritis may sometimes be an allergic reaction due to bacterial hypersensitivity, this has not been proved clinically, and there is little justification for vaccine therapy in cases of this condition.

TRACHOMATOUS KERATITIS

A BIOMICROSCOPIC STUDY OF TWO HUNDRED AND EIGHTY INDIAN SCHOOL CHILDREN

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Prior to the advent of the biomicroscope the pannus of trachoma was generally believed to be a frequent but not invariable complication of the disease, occurring usually months or years after the onset. The slit lamp studies of Dusseldorp,¹ Danilevskij,² Wilson,³ Cuénod and Nataf,⁴ Gallemaerts,⁵ Busacca,⁶ Horváth,⁷ Morax,⁸ Morax and Petit,⁹ Howard¹⁰ and others have indicated that pannus is not a complication of trachoma but an integral part of the disease, appearing in its earliest stages. Of particular interest have been the reports of Cuénod and Nataf from Tunis, Wilson from Egypt, and Busacca from Brazil.

Cuénod and Nataf found biomicroscopic signs of pannus in children with pure trachoma at a time when gross examination revealed no changes. Wilson, considering only the vascular changes of pannus, found extension of capillary loops into the cornea invariably in Egyptian trachoma. In patients followed from the onset he observed that the first changes of pannus usually appeared simultaneously with the

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1. Dusseldorp: Tracoma, Thesis, National University, Buenos Aires, 1927; cited by Cuénod and Nataf.⁴

2. Danilevskij, I.: Ueber die Bedeutung der Spaltlampe zur Diagnostik des Trachoms, *Zentralbl. f. Ophth.* **24**:641, 1930.

3. Wilson, R. P.: A Short Slit-Lamp Study on the Corneal Vessels in Egyptian Trachoma, *Folia ophth. orient.* **1**:52, 1932.

4. Cuénod, A., and Nataf, R.: Biomicroscopic de la conjonctive, Paris, Masson & Cie, 1934, p. 90.

5. Gallemaerts: Trachome et coloration vitale, *Ann. d'ocul.* **161**:815, 1924; Examen microscopique des affections de la cornée au moyen de la lampe à fente, *Bull. et mém. Soc. franç. d'ophth., supp.*, May 1926.

6. Busacca, A.: La frequenza della complicazioni corneali nel trachoma de dotta dallo esame de cento casi, *Rev. internat. du trachome* **10**:57, 1933; Recherches sur l'étiologie du trachome et sur la localization du trachome a la cornée, *Rev. biol. e hyg.* **5**:88, 1934; Die trachomatöse avaskuläre Keratitis, *Klin. Monatsbl. f. Augenh.* **94**:202, 1935.

7. Horváth, B.: Hornhaut bei Trachom, *Klin. Monatsbl. f. Augenh.* **72**:243, 1924.

8. Morax, V.: Complications cornéennes du trachome, *Rev. internat. du trachome* **6**:90, 1929.

9. Morax, V., and Petit, J.: Le trachome, Paris, Jean Morax, 1929.

10. Howard, H. J.: The Diagnosis of Trachoma, *Am. J. Ophth.* **16**:132, 1933.

conjunctival lesions. In cases of follicular and catarrhal conjunctivitis he found no vascular changes. Busacca considered the trachomatous cornea in detail, controlling his slit lamp studies with microscopic examination of material removed for biopsy. This control enabled him to correct certain errors made by previous investigators as to the vascularization of the normal limbus, the nature of the so-called palisades and the occurrence of trachomatous follicles in the cornea. He found avascular keratitis in all the cases of active trachoma which he observed and noted that it occurred before vascularization in patients followed from the onset.

A previous report¹¹ recorded the occurrence of pannus in a small series of patients with trachoma in the white population of Iowa and differentiated trachomatous pannus from the pannus associated with other diseases. It was concluded that biomicroscopic examination of the region of the upper limbus was of particular value in distinguishing cases of early trachoma from those of inclusion conjunctivitis in which pannus does not occur. This is the disease most commonly confused with trachoma in this country.

In view of the divergence of opinion among physicians of the Indian Medical Service and visiting ophthalmologists as to the frequency of pannus among trachomatous Indian children, it seemed desirable to undertake a slit lamp study of a representative series of Indian patients. Permission was received from the Office of Indian Affairs to examine infected children at the school for children with trachoma, Fort Apache, Ariz., and normal children from the school at Whiteriver, Ariz. Dr. F. I. Proctor, consultant on trachoma, and Dr. James C. Hancock, special physician, both of the Indian Medical Service, gave personal assistance in these studies, and Dr. Polk Richards, director of trachoma activities, gave help and advice.

MATERIAL AND METHOD OF STUDY

Two hundred and forty-eight Indian children varying in age from 6 to 19 years were available for study. All had been placed in the school because of trachoma or suspected trachoma and were receiving appropriate therapy. In addition, 32 children from the Whiteriver school, selected because of normal conjunctivae, were observed as controls.

Except for corneal scars suggestive of previous phlyctenular keratitis in 3 children, no evidence was found of other corneal disease capable of producing pannus. According to Dr. Richards, phlyctenular keratoconjunctivitis was formerly common but decreased in frequency after increased appropriations for food in the boarding-schools. Only a single case of active phlyctenulosis was observed in the two schools during a thirty day period of observation.

Routine examination at a magnification of 22 diameters was made of the entire cornea of each child; special attention was given to the region of the upper limbus.

11. Thygeson, P.: Biomicroscopy of the Limbus Corneae in Trachoma and Other Conjunctival Diseases, *Am. J. Ophth.* **17**:787, 1934.

REPORT OF OBSERVATIONS

Characteristics of Trachoma in the Indians.—Trachoma as observed in the Apache Indians appeared to differ in no essential way from trachoma in the white population. The lesions presented by the children of this series could be classified readily according to MacCallan's¹² classification; all stages of the disease occurred. While symblepharon, severe scarring and gross pannus were observed in the children, the complications of trichiasis and entropion were seen only in the adult population. Examination of smears of secretion and epithelial scrapings indicated that the incidence of secondary bacterial infection was higher than that in trachoma in the white population of Iowa. The most common bacteria found were *Haemophilus lacunatus* (Morax), *Haemophilus influenzae*, *Diplococcus pneumoniae* and *Staphylococcus*. Halberstaedter-Prowazek inclusion bodies were present in a considerable number of cases.

The Normal Limbus.—The limbus of the Indian children, except for the presence of considerable pigment in the epithelium, showed no peculiarities not found in white persons. In general, palisade formation was inconspicuous. As in the previous study, the semiopaque border of the limbus as viewed by focal illumination was found to be a satisfactory landmark by which to judge vascularization. In the 32 normal children the end capillary loops of the limbus extended to, or slightly beyond, this border.

The Trachomatous Limbus and Cornea.—The changes of trachomatous pannus were observed in all but 8 of the 212 children whose condition was diagnosed as trachoma on the basis of the conjunctival lesions alone and in 16 of the 36 whose condition was classed as "doubtful trachoma." Seven distinct lesions were observed: (1) extension of vessels into the cornea; (2) limbic follicles; (3) Herbert's peripheral pits; (4) diffuse subepithelial infiltrates; (5) localized subepithelial infiltrates; (6) minute punctate epithelial lesions, stainable with fluorescein; (7) corneal scars. So-called pustules of trachoma, which, according to Busacca, are collections of polymorphonuclear leukocytes, were not observed in the children but were seen in a single adult Indian with acute exacerbation of old trachoma. They have been observed not uncommonly in cases of trachoma in the white population.

Vascularization.—There appeared to be an extension of preexisting limbic loops into the cornea rather than formation of new blood vessels. The degree of penetration varied from about 1 mm. at the upper limbus to complete vascularization of the cornea. In mild pannus only the upper two fifths of the cornea was involved, but if the pannus

12. MacCallan, A. F.: *Trachoma and Its Complications in Egypt*, London, Cambridge University Press, 1913.

was at all severe there was penetration into the cornea over its entire circumference, always more extensive above.

Although pannus is usually classified as incipient, active or cicatricial, it seemed desirable for the purposes of comparison to classify it according to the extent of penetration of the vessels. Thus, four degrees of pannus were recognized, as follows: In first degree pannus the vessels extended approximately 1 mm. into the cornea at the upper limbus (fig. 1 *A*); in second degree pannus, approximately halfway to the pupillary area (fig. 1 *B*); in third degree pannus, approximately to the edge of the pupillary area (fig. 1 *C*), and in fourth degree pannus, over the entire cornea (fig. 1 *D*). The caliber of the vessels appeared to vary directly with the activity of the pannus: In cicatricial or healed pannus the vessels were small and in many instances contained no blood, whereas in pannus of high activity with circumcorneal congestion the vessels were large and dilated.

Limbic Follicles.—Limbic follicles (fig. 1 *D*), varying in number from three to thirteen, were present in 12 of the 204 patients with pannus. They were grayish, translucent globular masses, avascular except for occasional fine capillaries ramifying on their surfaces. They were arranged usually in a single row, concentric with the cornea, with the largest in the center, but were occasionally in double rows. In this series they were not seen below the horizontal meridian. They occurred most frequently in third degree pannus but were also present in second degree pannus and fourth degree pannus. They were never seen in the cornea itself. The description of trachomatous follicles in the corneal tissue by some observers has probably been the result of confusion of trachomatous pustules with trachomatous follicles. According to the histologic studies of Busacca,⁶ the pustules are made up almost entirely of neutrophil leukocytes and are a manifestation of acute corneal trachoma. The rupture of the epithelium over the pustules, with the formation of shallow ulcers, is therefore in no way comparable to the rupture of trachomatous follicles in the conjunctiva.

Herbert's Peripheral Pits.—Typical pits, the remains of limbic follicles which have undergone absorption, were observed in 11 of the 204 patients with pannus. Like the follicles, they were usually bilateral but in a few instances were seen in only one eye. They appeared as round or oval semitransparent areas (fig. 1 *C*) in the white opacity of the limbus. They were usually arranged in a single row, with the largest in the center. Unlike the limbic follicles, which bulged forward, they appeared as flat or depressed areas. The term pit, however, would seem to be misleading since there is no actual depression. When not marked they gave the limbus a grossly fenestrated or arcade-like appearance.

Diffuse Subepithelial Infiltrates.—Diffuse infiltrates were seen frequently, especially in the region of new vascularization. In some instances this infiltration was so dense as to render determination of the semiopaque border of the limbus difficult.

Localized Subepithelial Infiltrates.—Localized infiltrates were especially well seen in the vessel-free parts of the cornea. They varied in size and number but were usually irregular in shape, sometimes having pseudopodia-like processes. In some cases they could be observed undergoing apparent replacement by scar tissue.

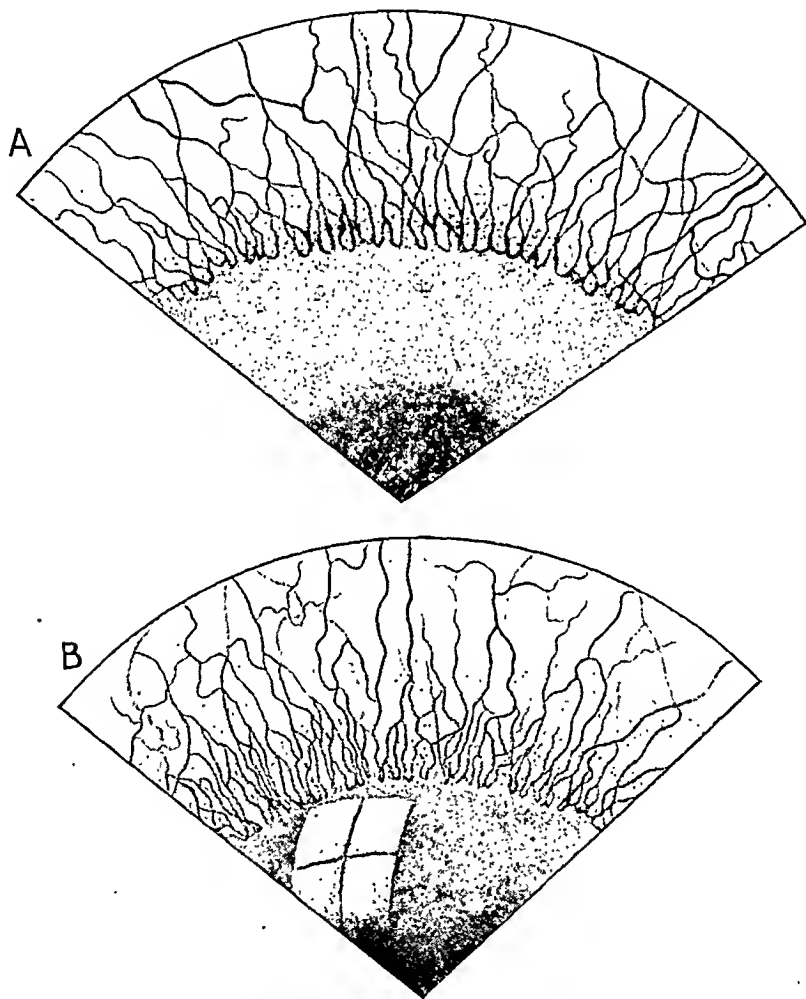


Fig. 1.—*A*, first degree pannus in an Indian child with trachoma of stage I (incipient trachoma). Capillary loops extend into the cornea well beyond the normal border of the limbus. Beyond the vessels are spotty infiltrates. The normal sheen of the cornea is reduced by edema of the epithelium and by punctate epithelial lesions which stain with fluorescein. *B*, second degree pannus in an Indian child with trachoma of stage III (cicatricial trachoma). The vessels extend about half-way to the pupillary area. The normal border of the limbus is somewhat obscured, owing to infiltrates. There is definite keratitis beyond the vessels, evidenced by infiltrates, by edema of the epithelium and superficial layers of the corneal stroma and by punctate epithelial lesions.

Epithelial Lesions.—Almost every patient showed the minute punctate epithelial lesions (fig. 2) which stain with fluorescein. These varied greatly in extent, in general being in proportion to the activity of the pannus as judged by infiltration, dilatation of the vessels and circumcorneal congestion. All the vessel-free parts of the cornea were involved in one or another of the cases, but the region of the pupil and the area just above it were most commonly affected.

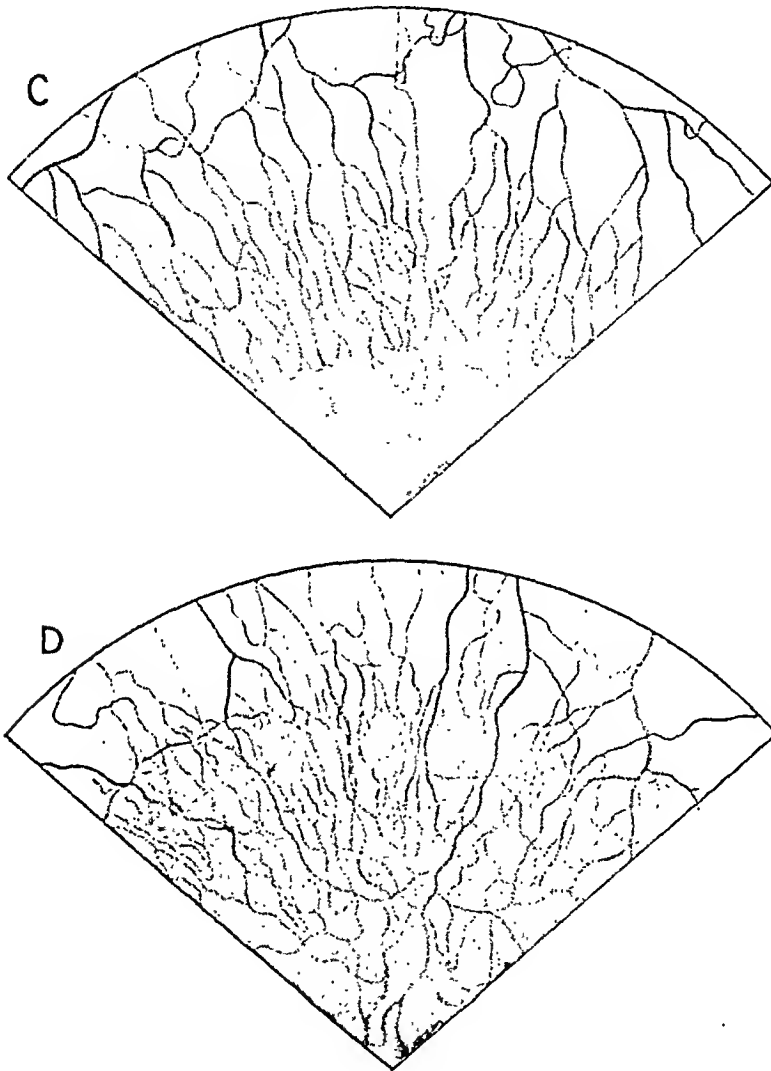


Fig. 1.—C represents third degree pannus in a child who suffered from trachoma of stage III. The vessels extend to the pupillary area and are of coarse mesh. In addition to active infiltrates, epithelial lesions and corneal edema, there are a number of round scars, probably evidence of healed ulcers. At the limbus are Herbert's pits, the remains of previously existing limbic follicles. D, fourth degree pannus in association with trachoma of stage III. The entire cornea is vascularized. The continued activity of the corneal disease is evidenced by dilated vessels, infiltrates and limbic follicles.

Corneal Scars.—Scars resulting from previous ulceration were seen in only a small number of cases. In some instances of extensive pannus cicatricial tissue had replaced subepithelial infiltrates.

Correlation of Extent of Pannus with Stage of Trachoma.—In table 1 the degree of pannus is correlated with the stage of trachoma. In trachoma of stage I, as would be expected, first degree pannus was twice as frequent as second degree pannus. There was one case of third degree pannus, showing that severe pannus can occur prior to the onset of conjunctival scarring and relatively early in the disease. In trachoma of stage IIa first degree pannus and second degree pannus were the rule, but there were 2 cases of third degree pannus. In trachoma of stage IIb, first degree pannus and second degree pannus were about equally common. In trachoma of stage III all degrees of pannus were present, 29 of a total of 134 patients having the extensive vascularization of third degree pannus. There were only 2 cases in which the cornea was completely vascularized. It is interesting that in 8 cases¹³ of healed trachoma, pannus was only of the first or the second degree.

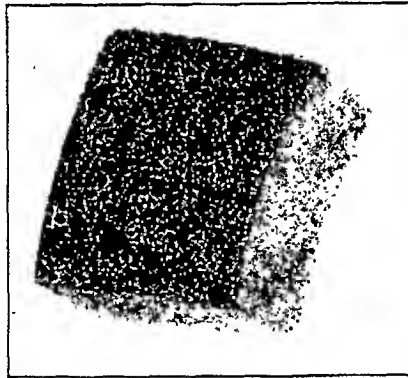


Fig. 2.—Minute punctate epithelial lesions, which stain with fluorescein, in the vessel-free part of the cornea in a patient with extremely active pannus.

TABLE 1.—*Extent of Pannus and Stage of Trachoma*

Stage of Trachoma	Extent of Pannus*				Total
	First Degree	Second Degree	Third Degree	Fourth Degree	
I.....	7	4	1	0	13
IIa.....	18	12	2	0	32
IIb.....	12	11	0	0	23
III.....	57	46	29	2	134
IV.....	1	1	0	0	2
					<hr/> 204

* See figures 1A to D. When the degree of pannus differed in the two eyes, the more advanced degree was recorded.

Correlation of Extent of Pannus with Age.—As indicated in table 2, there was no sharp correlation between the age of the child and

13. Six of these cases were instances of healed trachoma observed in the Whiteriver school.

the degree of pannus. In general, however, the relative frequency of second degree pannus and third degree pannus increased with age; complete vascularization (fourth degree pannus) was observed in only 2 patients, both aged 14 years.

Pannus in Doubtful Trachoma.—Pannus was present in either the first or the second degree in 16 of 36 children with a condition classified as doubtful trachoma on the basis of conjunctival examination.

None of the patients had scarring or characteristic lesions of the upper tarsal conjunctivae, and it would have been impossible by gross examination alone to make an indisputable diagnosis of trachoma. The occurrence of follicles at the upper border of the tarsus and in the upper fornix, however, was suggestive of trachoma, and in the presence of the vascular and infiltrative changes of early pannus was sufficient to establish a diagnosis.

TABLE 2.—*Type of Pannus and Age of Child*

Age, Years	Extent of Pannus*				Total
	First Degree	Second Degree	Third Degree	Fourth Degree	
6.....	4	1	0	0	5
7.....	4	3	4	0	11
8.....	16	9	0	0	25
9.....	10	8	2	0	20
10.....	11	5	2	0	18
11.....	10	6	1	0	17
12.....	6	3	4	0	13
13.....	9	12	5	0	26
14.....	8	7	4	2	21
15.....	8	7	4	0	19
16.....	5	9	1	0	15
17.....	4	2	2	0	8
18.....	4	1	0	0	5
19.....	1	0	0	0	1
					204

* See figures 1A to D.

Significance of Trachoma Without Pannus.—In 7 of the 8 cases in which the condition was diagnosed as trachoma but in which no pannus could be discovered the changes were those of stage 3 and showed little, if any, activity. In view of the fact that many of the children had been previously subjected to grattage it is not impossible that the scars on the basis of which the diagnosis of trachoma was made were mechanical and not trachomatous. The eighth patient showed no scars and, except for the absence of involvement of the upper tarsal conjunctiva, had the follicular hypertrophy of stage IIa. The predominance of involvement of the lower lid was suggestive of inclusion conjunctivitis.

In none of the 8 cases was there avascular keratitis.

COMMENT

The observations just reported indicate that pannus occurs early in the course of trachoma in Indian children and that slit lamp examina-

tion of the limbus and cornea is of diagnostic value in doubtful cases. It is recommended, therefore, that biomicroscopic examination of the limbus and cornea be made as a routine in the examination of patients suspected of having trachoma. It is believed that the consistent use of the slit lamp will simplify the differentiation of early trachoma from follicular and other types of conjunctivitis.

There is no doubt that pannus occurs frequently in association with trachoma, but whether or not it invariably accompanies the disease is not yet satisfactorily established. Slit lamp examination of patients with trachoma in the white population during the past four years has always shown pannus, although the changes were mild in some instances. In several cases, in spite of pronounced conjunctival lesions, gross pannus never developed, although permanent biomicroscopic signs of previous disease persisted at the limbus even after healing. At the other extreme was a case in which pannus was grossly visible six weeks after the onset of the symptoms and in which, in spite of treatment, vessels reached the pupillary area within twelve months. The constancy of corneal involvement in trachoma of the white population favors the view that it always occurs in association with the disease and that the 8 children with normal corneas in the series of Indian patients were therefore not trachomatous.

SUMMARY AND CONCLUSIONS

1. The vascular and infiltrative changes characteristic of trachomatous pannus were observed on biomicroscopic examination in 203 of 211 Indian children with a condition diagnosed as trachoma on the basis of the conjunctival findings alone, a percentage of 96.2 per cent.

2. Seven of the 8 children without pannus had scarred conjunctivae with minimal inflammatory changes. It is possible that in these cases the condition was not trachoma but follicular conjunctivitis which had been subjected to grattage at some previous time. The eighth child had changes suggestive of inclusion conjunctivitis.

3. The important biomicroscopic signs of trachomatous disease of the cornea are briefly discussed.

4. Limbic follicles or their remains, Herbert's peripheral pits, were found in approximately 20 per cent of the patients with pannus.

5. Pannus was seen in 16 of 36 children with a condition classed as doubtful trachoma.

6. Pannus was not observed in 32 children with normal conjunctivae. The cicatricial remains of pannus were present in 8 children with healed trachoma.

7. This study supports the conclusion that the first signs of trachomatous pannus appear early in the disease.

PHYSICS OF DIATHERMIC COAGULATION IN THE EYE

EXPERIMENTAL STUDIES AND SOME PRACTICAL NOTES ON THE OPERATION

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The best known method for healing retinal detachment is diathermic coagulation. Nearly every ophthalmologist has his special method for carrying out diathermic operations. All these methods lack a reliable control of the dosage, because the physical basis of diathermic coagulation has been neglected.

My co-worker, Mr. Heinrich Kalmus, and I have found a convenient method of observing the process of coagulation in the measurement of the electrical resistance of the eye. From the variations of the resistance reliable conclusions may be drawn as to the state of coagulation.

In this article adequate test methods and the results of experiments are described. On the basis of these experiments the relative values of the different methods of operation are considered. The knowledge of electrical resistance furnishes a convenient and accurate method to indicate the proper dosage of diathermic irritation in the human eye.

SURVEY OF THE PREVIOUS LITERATURE

The widespread treatment for healing retinal detachment is diathermic coagulation. The methods of operation, however, as suggested by various authors are quite different. Some prefer surface coagulation; others use micropuncture or a combination of the two methods.

It is sufficient to mention one exponent of each of these methods to show how different the opinions are. Coppez¹ maintained that surface coagulation leads to adhesive choroiditis, which is the principal condition of healing, whereas the use of pins may cause grave infiltration and damage of the retina. Vogt,² on the contrary, expressed the belief that surface coagulation has the disadvantage, common to all surface methods, of leading to diffuse chorioretinitis, thereby damaging larger retinal parts than is necessary. The atrophy of the retina hinders

From the Department of Ophthalmology, the Jewish Hospital, Budapest, 1935.

1. Coppez, Léon: *Arch. internat. de méd. expér.* **9**:177, 1934.

2. Vogt, A.: *Arch. f. Ophth.* **133**:26, 1935.

ophthalmoscopy, and therefore holes may remain unnoticed. Repeated operations will be difficult partly because of the reason already mentioned and partly because of the solid adhesions between the eyeball and the surrounding tissue. Larsson,³ Shapland⁴ and others, however, have reported satisfactory results obtained with the method of surface coagulation.

Šafář's⁵ pins are short, being just long enough to pierce the sclera, and his excellent results seem to justify his method. Walker⁶ uses pins too, desiring to obtain a combined effect. He carries out micropuncture with the tip of the pin, while an electrical nonisolated stopping plate of the needles causes surface coagulation on the sclera. The combined method is used also by Weve,⁷ who first produces surface coagulation with a blunt electrode and then carries out micropuncture with short pins. In the case of cystlike detachments, when the retina is bulged deep into the vitreous, Weve often uses pins 3 to 4 mm. long. Though this procedure may sometimes be rather dangerous, as the retina may be damaged by the tip of the pin, yet he seems to have obtained most excellent results with this method.

Opinions differ as to the piercing depth of diathermic pins. Meesmann⁸ expressed the belief that the effect of diathermic coagulation of the retina is very beneficial. He runs the pin close to the retina and uses a current of an intensity and duration just sufficient to produce a small gray-white focus on the retina.

A healing effect from the diathermic current can be expected only if the dose is kept within definite limits; underdosage or overdosage may do harm. Ophthalmologists therefore endeavor to check the dosage. As the heat is due to the diathermic (electric) current, it might seem that the dosage could be controlled by measuring and adjusting the intensity, i. e., the milliamperes, of the diathermic current. This method has been proposed by different authors, yet their experience was that while they used the same milliamperage and disregarded some other circumstances different degrees of coagulation resulted. Obviously, coagulation depends not only on the intensity of the diathermic current but also on many other factors, such as the humidity of the sclera and the electrical resistance of the tissue.

3. Larsson, Sven: *Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **50**:113, 1935.

4. Shapland, C. D.: *Brit. J. Ophth.* **18**:1, 1934.

5. Šafář: *Behandlung der Netzhautabhebung mit multipler diathermischer Stichelung*, Berlin, S. Karger, 1932.

6. Walker, C. B.: *Am. J. Ophth.* **17**:1 (Jan.) 1934.

7. Weve, H.: *Ann. d'ocul.* **171**:1, 1934.

8. Meesmann, A.: *Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **50**:121, 1935.

Though opinions differ regarding the various modes of diathermic operation, most authors acknowledge that as yet diathermy is the most suitable way for healing retinal detachment. However, Vogt has stated that diathermy is dangerous and should be abandoned. He is looking for a new method and thinks that he may find it in electrolysis, which was applied first by Imre⁹ and later by von Szily and Machemer.¹⁰ His arguments against the use of diathermy are: (1) A very high temperature (more than 1,000 C.) is produced on the site of the puncture, due to sparks, which may easily occur at the customary intensity of from 50 to 120 milliamperes, and (2) in case the tip of the pin penetrates deeper into the eye it may remain cold; the detached retina may be punctured without the production of coagulation, and new detachment will ensue.

Vogt's objection to diathermy lent a special interest to research in the physics of diathermic coagulation. Apart from this the question would have arisen, as complete control of the dosage could not be based only on the ground of the knowledge of the physical happenings during the process of coagulation. Investigations concerning diathermic coagulation have been made hitherto mostly from the histologic point of view. Only a few of them dealt with the physical process; e. g., Coppez in his experiments observed the distribution of diathermic heat and endeavored to find a relation between the different factors of diathermic coagulation and that of the heat produced.

I found in some tests that the electrical resistance of the eye is closely related to the state of coagulation. Further investigations proved that when diathermy is not applied the value of resistance is practically independent of the condition of the eye of the patient, and its variations during the operation show clearly the process of coagulation. My co-worker and I found an easy means of measuring the resistance during diathermic application and by this obtained some new light on diathermic coagulation.

A NEW METHOD OF MEASURING RESISTANCE IN TISSUE UNDER TREATMENT WITH HIGH FREQUENCY ENERGY, WHICH MAY BE USED AS A MEANS OF ADJUSTING DOSAGE

HEINRICH KALMUS

It was possible adequately to utilize the beneficent action of roentgen rays only after suitable apparatus for controlling roentgen dosage had been devised. Those who attempt to use high frequency currents (for surgical or other therapeutic procedures), diathermic currents or currents of any other similar type are confronted with the same problem of suitable apparatus. A new dosage is described here

9. Imre, J.: *Klin. Monatsbl. f. Augenh.* **89**:545, 1932.

10. von Szily, A., and Machemer, H.: *Klin. Monatsbl. f. Augenh.* **90**:806, 1933.

which, although originally developed for the special purpose of interference in ablatio retinae, may be utilized for a whole group of other diathermic operations. Thorough testings have been made with the collaboration of Dr. Klein.

The present day methods of dosage are so extremely unreliable that the successful outcome of an intervention has come to depend largely on the skill of the individual physician. The action of a high frequency current on the tissue has been measured solely in terms of the current's intensity as registered on a heated wire or thermocross instrument and by the duration of the application. The onset of coagulation may well be manifested by discoloration at the point of puncture but, as the appearance of this area is not altered by further action, no satisfactory conclusion can be drawn with reference to the effect exercised by the current—an effect which, as a result of the sparks given off, may inflict serious damage. Not long ago an attempt was made to improve the technic of dosage, but no complete solution of the problem has been effected thereby. This method substituted for the ordinary needle an instrument of puncture having two metals soldered together at its tip. The soldered area forms a thermal element that may be applied after the instrument has been connected with the apparatus and with a compensation cell which aids in the maintenance of a constant temperature of the room and of the tissue and may be placed in the immediate vicinity of the punctured center.

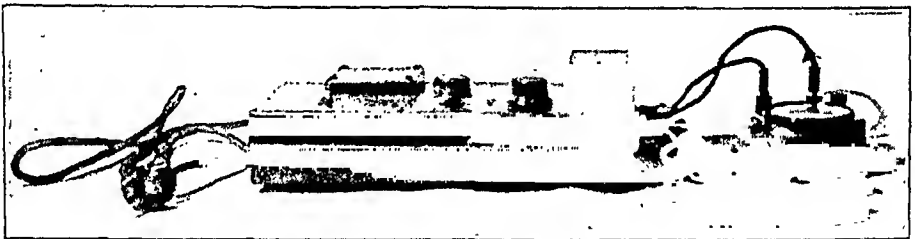


Fig. 1.—The measuring apparatus.

Unfortunately, the foregoing method has two disadvantages. In the first place, owing to the use of two metals soldered together, the point is less fine than that of a single, uniform needle. Secondly, the temperature of the center of puncture cannot be as quickly determined; the point acquires a lag of heating due to the length of time required for the heating process. Certain harmful aspects of the treatment with current, flying sparks for example, are not as a rule to be observed.

Our new apparatus provides for measurement of the ohmic resistance in the treated tissues and makes possible the constant observation of the tissue alterations throughout the operation. The heat of the high frequency current produces both desiccation, that is, reduction in the water content, and chemical alterations in the tissue. One such chemical change is the congealing of the protein which characterizes coagulation. Both desiccation and coagulation result in a marked change in the ohmic resistance and, from the observation of these phenomena, the effect of the high frequency current may be judged.

The resistance is determined by computation of the voltage. A steady measured current flows through the tissue together with the high frequency current and by this measured current the decrease in voltage which the current undergoes as a result of resistance is computed. Naturally, caution must be taken that no contrary influence on the circulation of the high frequency and measured currents is present.

It suggests itself as most probable that the measured current should be a direct current. Experimentation has shown, however, that owing to phenomena of polarization uncontrollable electromotive forces enter between the electrodes and prevent the accurate measurement of resistance. For this reason a 50 period alternating current was used, and a tubular voltmeter selected as indicator of the tension. This instrument should be insensitive to direct tension, and in this way the inaccuracies of the older methods will be eliminated.

The magnitude of the measured current is so restricted that it cannot provoke excitement of any kind. In numerous experiments with animals the maximal value permissible was 0.2 milliampere. For the dosing apparatus a value of only 0.1 milliampere was selected in the interest of safety.

The entire measuring system consists of three parts: a high frequency-measured current switch for separation of the two currents, the transformer and the instruments for supply and regulation of the measured current and finally the highly sensitive tubal voltmeter for measurement of the measured current and of the drop

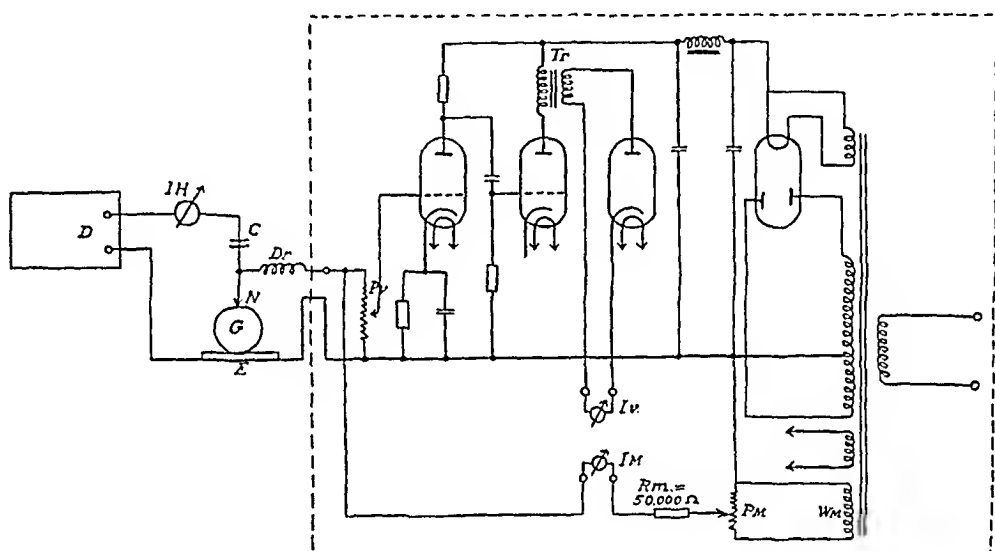


Fig. 2.—Construction of the measuring apparatus connected with the diathermic apparatus. The designated parts are: *D*, diathermic apparatus supplying high frequency current; *IH*, measuring instrument; *C*, condenser; *N*, operating needle; *G*, tissue (enucleated pig's eye) being treated; *E*, grounded plate; *Dr*, choke; *Pv*, potentiometer; *Tr*, transformer; *Iv*, ampere meter for diode current; *IM*, ampere meter for the alternating current (measured current); *RM*, fixed resistance; *PM*, potentiometer; *WM*, secondary winding for alternating current (measured current).

in potential in the tissue under treatment. With the exception of the switch, which must be connected directly with the operating needle in order to avoid loss of capacity in the high frequency current, the other parts are attached to a compendious apparatus connected with the electric current and easily so constructed as to form one unit with a diathermic apparatus.

The complete arrangement of the measuring apparatus is to be seen in figure 2. The section of switch bounded by the broken lines is built into the aforementioned apparatus.

The diathermic apparatus *D* supplies the high frequency current which flows by way of the instrument *IH*, the condenser *C*, the operating needle *N* to the tissue (in the experiments described in this paper, an enucleated pig's eye) under treatment, then through inactive electrode *E* back to *D*. Coil *WM* of the current transformer supplies the measured current. The potentiometer *PM* serves to

regulate the tension. By this procedure the measured current is made to flow by way of the resistance RM , a milliamperemeter IM and a high frequency coil of great impedance Dr (which weakens or suppresses alternating currents) into the operating needle. Thence it flows through the tissue under treatment back again to the transformer by way of the inactive electrode. The magnitude of the measured current is maintained constant by the resistance RM , the value of which is many times greater than that of the tissue resistance, some 50,000 ohms against 5,000 ohms. Therefore, the drop in potential suffered by the measured current in the tissue is directly proportional to the tissue resistance. The high ohmic potentiometer Pv is attached to the coil of impedance parallel to the tissue. Some of this tension is exhausted at Pv and conducted to the screen of the first tubes. These tubes function as strengtheners of resistance. From the anode an increased potential passes through a condenser and the screen of the second tubes, is here further strengthened and finally flows through a transformer to a diode, where it is rectified and measured with an ordinary rotary spool. The transmission-transformer between the tubes and diode functions so perfectly that the greatest possible diode resistance is adjusted favorably to the tube resistance, and the tubes may function to capacity.¹¹

The new dosage method assures more favorable results from the use of high frequency currents in operative interventions. In many situations in which the point of puncture is not at all visible (in operation on the trigeminal nerve, for example) the new apparatus makes possible, above all, a calculation of the magnitude of a current and the time required for its application quite independent of the physician's private judgment.

Figure 4 depicts a dosing apparatus with the mechanisms for the regulation of measured current and diode current.

[It should also be mentioned that a loud-speaker is connected with the rectified current, which gives a crackling tone if sparking occurs; this takes place when the coagulated spot becomes dry, and at the same time a sudden change of resistance can be observed. The loud-speaker signals the presence of sparks long before they could be detected by the eye.—M. K.]

RESULTS OF TESTS

The experiments made with the new apparatus described by Mr. Kalmus in the preceding section were expected to yield information about the following points: (1) the physics of coagulation and (2) the value of the different methods and theories of diathermic operation described in the ophthalmic literature.

1. *Physics of Coagulation.*—In the experiments my co-worker and I examined the methods that are commonly employed in operations, namely, (a) micropuncture and (b) surface coagulation.

(a) *Micropuncture (Microcoagulation):* As has already been mentioned, there are several factors which influence coagulation. There would be no use in changing all of them at the same time, but some variables should be kept constant during the test, and others should be changed. In our experiments we kept all the factors constant except

11. Mr. Kalmus, engineer, worked out this method of measuring resistance and constructed the apparatus for this purpose, and the engineers Mr. Márkus and Mr. Stern assisted in the performance of the experiments.

the diathermic current and the electrodes. Through this arrangement the relation of the diathermic current and the resistance of the eye could be exactly determined. The characteristics of the variation of resistance with time can be determined by means of measurements with different diathermic currents.

The pins used were provided with a stopping disk of ruby, which is a good isolator. They had a thickness of 0.15 mm. and a length of 1.8 mm.

After piercing the sclera with the pin we first measured the resistance of the "cold" eye, and afterward we connected the diathermic current. The resistance decreased immediately after the diathermic current was introduced into the eye and remained on this lower level for some time. When coagulation was finished the coagulated area became dry, and the electrical resistance increased steeply and reached a value which was very high in proportion to the initial value. The

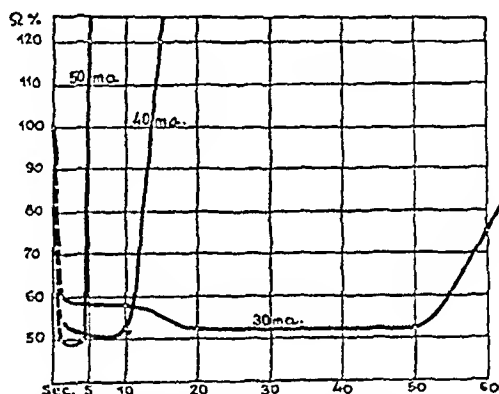


Fig. 3.—Variation of resistance with time for different values of high frequency current measured with needle electrodes.

part of the curve for the variation of resistance with time in cases in which the resistance remains low is the shorter the higher the intensity of the diathermic current applied (fig. 3). Though the variations in resistance depend largely on the circumstances of the test, it is expected that similarly shaped curves with changed values for the variation of resistance with time will be found if the circumstances are altered.

The results of the tests in detail are as follows:

In a group of the tests we applied 20 milliamperes of diathermic current; the initial resistance was 5,000 ohms. Under the influence of this current the resistance fell first to 3,500 ohms and then to 3,000 ohms, where it remained. We watched the eye as long as from forty to one hundred and seventy seconds after the connection of the current. The resistance was practically constant during this time. On the site of coagulation some scarcely visible alterations of the sclera took place, without drying up the coagulated area, in which case the resistance would obviously have risen.

Next we increased the current to 30 milliamperes. For instance: The initial resistance was 4,500 ohms. During coagulation the resistance fell to 2,400 ohms first and then became constant at 2,100 ohms for a time. Within from sixty to seventy-five seconds after the start the coagulated area dried up, and the resistance rose accordingly to 3,000 ohms and further to 7,000 ohms. Continuing the observation, in some tests we found values above 50,000 ohms. If one compares the results of this test with those of the former one, one sees that the difference is but quantitative. In the latter test the coagulation was more marked and was accompanied by the drying up of the coagulated area and the rise of resistance as shown in the curve.

Then we increased the current to 40 milliamperes. The initial resistance of 4,500 ohms soon fell to 2,100 ohms, and coagulation reached the period of drying up more rapidly. The resistance began to rise in about from ten to fifteen seconds.

With a further increase of the current of about 50 milliamperes coagulation took place in from three to five seconds, and if the current was adjusted to any value above 50 milliamperes coagulation took place so rapidly that it could not be observed. This means that the applied current must not exceed the proper value (in the test in question, about 30 milliamperes), else overdosage and consequent harm to the eye may occur.

During the tests just described in the period of drying up we could hear continuous crackling by the loud-speaker, signaling the presence of sparks, though no spark could be detected by the eye. The loud-speaker proved very useful in these tests, as sparks were signaled at the moment of their development.

In analyzing the characteristics just described four periods can be distinguished:

1. A period of rapid fall of the resistance, which occurs as soon as the diathermic current is connected. This part of the curve was drawn on the charts with dotted lines. Owing to the suddenness of the drop in resistance, the exact relation of resistance and time could not be noticed.

2. A period of a steady state of resistance on a low level. This period lasts till the coagulated area becomes dry.

3. A period of a sudden rise of the resistance. This is the period of thorough drying up. In this interval small sparks usually occur. They are invisible but are detected by the loud-speaker.

4. A period of carbonization of the tissue. In this period of coagulation the resistance rises to extremely high values, and damage of the tissue is conspicuous, though the degree of destruction cannot be easily estimated.

It is hard to judge whether the observed sparks are harmful or not. The damaged part of the tissue can be considered rather negligible in relation to the bulk of the eye. It is a question, however, whether such a delicate organ as the eye can suffer such injury, even on a small area, without danger. From the purely physical point of view one can state that in the presence of sparks control over the high frequency current is made impossible.

The tests already described have been made repeatedly, and the process of coagulation was nearly similar in each case at any of the adjusted milliamperes of current. The results can therefore be reproduced. It must be stated, however, that a similar process of coagulation can be obtained in such cases only when the circumstances of the test, i. e., those of the operation, are standardized, because both the process and the degree of coagulation depend on such circumstances as the milliamperage, the electrodes and the depth of piercing.

(b) Surface Coagulation: The arrangement of the experiment described under *a* was kept unchanged except for the pins, which were replaced by blunt electrodes 0.8 mm. in diameter. If the sclera is dry the initial resistance of the eye measured by means of such an electrode is rather high. After the diathermic current is switched on, even with a current of not more than 20 milliamperes, the resistance rapidly rises to values so high (over 50,000 ohms) that measurement is difficult. The explanation of this fact is that a thin film of scorched tissue develops rapidly on the dry sclera, and this film is a bad conductor. Soon sparks of visible intensity develop, and at the same time the milliammeter shows a drop of the current sometimes nearly to zero. That is a consequence of the efficient rise of resistance.

Most authors suggest the use of a current of about 100 milliamperes. If one did not take into consideration the mentioned change of current and persisted in using the proposed intensity of 100 milliamperes by readjusting it with the regulating knob, the output of the diathermic apparatus would soon exceed the limits of that for a surgical operation. The milliammeter is useful in this case too, since by showing the rapid drop of current it gives some indication as to the state of coagulation and prevents one from readjusting the current, which would lead, of course, to overdosage. One learns from the process described that no full information can be obtained regarding coagulation by the use of the milliammeter alone, however useful this instrument may be.

Frank Flynn's method proved very good, as was demonstrated by further experiments. According to this method the sclera is moistened with distilled water all the time during surface coagulation. After the connection of the current the behavior of the resistance is similar to that we have seen when pin electrodes have been used; that is, after a

rapid fall the resistance remains permanently low. As the continuous moistening of the sclera prevents the area of coagulation from drying up, the rising part of the curve is missing. Some results of this test at different intensities of current are as follows:

With a current of 50 milliamperes, after the connection of the current the initial resistance was suddenly reduced by from 12 to 14 per cent, and during sixty seconds' observation a slight, hardly visible area of coagulation had formed. No shrinking could be detected.

With a current of 70 milliamperes the process was similar, but the change in the color of the sclera was more conspicuous, and in about thirty seconds shrinking could be observed.

With a current of 80 milliamperes the drop of resistance was from 18 to 20 per cent, and its value remained constant for the observation time of sixty seconds. The sclera was characterized by rapidly growing change of color, coagulation and marked shrinking. If coagulation went on, moistening was no longer able to prevent shrinking of the sclera. This shrinking was the more intensive the greater the applied current. At a current of 100 milliamperes shrinking took place suddenly (in from two to four seconds). With a very strong current it was impossible to prevent sparks and carbonization by the usual moistening.

It seems from this series of tests that if the sclera is dry it is rapidly scorched by surface coagulation. The carbonized layer then prevents coagulation from spreading into deeper layers; besides sparks develop which may harm the eye.

If, on the contrary, the coagulating spot is moistened, carbonization is avoided. This method might be called endothermic, because the generated heat is propagated into the deeper layers of the eye too. With the aid of this method coagulation seems to have a greater effect on the choroid. It can be concluded from the foregoing observations that surface coagulation must not be employed without moistening the sclera. It must be mentioned, however, that only distilled water may be used for moistening, since salt components or serums render the electrical resistance of water too low.

2. Methods Mentioned in the Literature.—Some differences are found in the literature regarding the features of the operation. By further experiments, which are described in the following paragraphs, one is able to compare the merits of the different methods and their principles.

Some ophthalmologists lay stress on keeping the sclera dry, if the operation is carried out with pin electrodes. The liquid film on the eye is supposed to lead away the high frequency current and thus weaken the effect of coagulation. Our arrangement enabled us to solve this problem. Should the liquid film cause leakage the electrical resistance

would fall. So we pierced the sclera with the same pins as were used in previous experiments and measured the resistance. Then we dropped physiologic solution of sodium chloride on the sclera, but there was no change in the resistance. This is easily understood, considering that the part of the pin in contact with liquid film is small in proportion to the part pierced into the sclera (fig. 4). In case the liquid covered the isolating stopping plate and brushed the upper part of the pin (as is shown in figure 5) the expected drop in resistance took place. The same observations could be made after connecting the diathermic current. If one drops physiologic solution of sodium chloride on the sclera during the period of low resistance the resistance does not change unless the solution reaches the upper part of the pin or if the isolation of the stopping disk is damaged (e. g., if the enamel of the metal disk is peeled

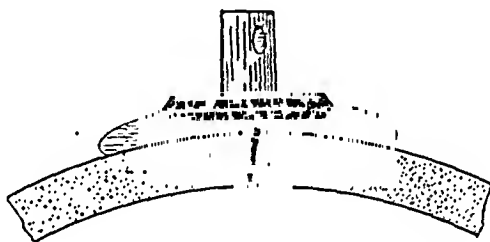


Fig. 4.—Diagram showing pin in contact with the liquid film and piercing the sclera.

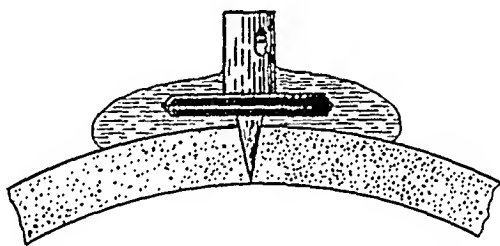


Fig. 5.—Diagram showing the liquid film covering the stopping plate of the pin.

off). The observations reported in the literature that similar coagulation can be obtained only if the sclera is kept dry seem to be due to such stopping plates, which enable the liquid film to touch a larger conducting surface.

It is important to learn what occurs when the pin is pierced through the fibrous shell of the eye and is pushed into the aqueous or vitreous part. We made the experiment with a longer unisolated pin, piercing as deep as 5 or 6 mm. The initial resistance was rather low. At a current of from 4 to 6 milliamperes no coagulation could be detected during a considerably longer time. If the current was increased up to 100 milliamperes coagulation developed in the neighborhood of the pin in the sclera as well as in the cornea, but practically no warming up of the deeper layers could be detected.

If the experiment is carried out in the way in which such operations used to be performed, i. e., if the diathermic current is connected first and the pin is pricked afterward, the process of coagulation is similar to the process that has already been described as long as the pin does not pass the sclera or the cornea. (This holds true in the case of a moderate current only.) However, as soon as the tip of the pin enters the vitreous coagulation ceases. Walker made similar observations. When the pin is in the aqueous or vitreous part it appears to behave like a thermocautery, which is hot in the sclera but cools down in the vitreous part. The main difference is that in the case of the thermocautery cooling is caused by leakage of heat, whereas in the case of the diathermic pins cooling is most probably caused by leakage of current through the vitreous, the resistance of which is lower than that of the sclera. As a result it may be stated that pins used in operation for retinal detachment must be equipped with a stopping plate to insure the proper depth of penetration. Besides, all pins used in ophthalmologic operations must be of the same thickness (the same surface) if the same effect is required with the same current.

There have been some attempts to use cutting current in coagulation recently; so we extended the investigations to this method. Even with a very low current, coagulation rapidly develops on the spot of piercing, and the resistance rises. The cause lies in the carbonization of a thin layer of the tissue touched by the pin. Thereby the resistance rises, and at the same time sparks develop. Cutting current therefore is unfit to be controlled either with a milliammeter or with any other device. Consequently the use of this method is not desirable.

THEORETICAL CONSIDERATIONS

Hitherto in the experiments described only two variables were investigated, namely, the electrical resistance and the intensity of the diathermic current. Other factors were left out of consideration by keeping them constant for each test. There are, however, many such factors which may influence the process of coagulation, and these should be considered when carrying out the diathermic operation. Information regarding these factors has been acquired partly by experience and partly by experiments. Investigations regarding these factors have been made by Weve, Coppez, Meesmann and other investigators, and it is well known today that the shape and size of the electrode, the piercing depth of the pin electrode, the specific heat and resistance of the tissue and the duration and frequency of the diathermic current have a marked influence on the development of coagulation. Jess suggested the use of high frequency current, though this should not be too

high because, according to his experience, current of lower frequency, is more suitable to produce coagulation. Weve has employed a frequency of 4,500,000 per second, but recently he informed me that lately he has been employing current of lower frequency.

In the choosing of frequency the most important point which should be considered is its strict relation to capacity current, which makes the determination of a proper dose difficult and may render the milliammeter useless for the control of the dosage at high frequencies.

D'Arsonval's experiment demonstrated clearly how much capacitive current may influence the results of the test. He placed in a glass jar two parallel plates of platinum as electrodes at a distance of 20 mm. apart, filled up the jar with solution of sodium chloride and changed the concentration of the liquid during the experiment to such an extent that he could change the resistance between these electrodes from 13 to 35,000 ohms. He connected high frequency voltage to the electrodes and observed the temperature of the liquid. The temperature naturally varied with the resistance, even if the current was kept constant. It was interesting, however, that this variation of temperature was not more than 8 C. when the variation in resistance was as large as from 1,500 to 2,500 ohms. The results obtained by d'Arsonval were correctly interpreted by Fabry, showing that the milliammeter indicates the sum of the currents passing through the ohmic resistance and the current. Capacitive current does not generate heat, and this part of the total current is the larger the higher the frequency. D'Arsonval's experiment cannot, however, be simply applied to the human body, because the opposite plate electrodes placed rather close to one another and the dielectric formed by the solution of sodium chloride increase the capacitive current far above that found in operations.

The uncertainty caused by capacitive current in regard to the dosage is further increased by the fact that the total current as read on the milliammeter is equal not to the arithmetical sum of the ohmic and capacitive currents but to the square root taken from the sum of the squares only. One may conclude that for not very high frequencies the reading of the milliammeter fairly approaches the actual value of the useful current.

A further argument against the control of the dosage by the milliammeter is the individual difference between the resistance of the eyes of different persons, the generated heat being proportional to the resistance. We measured the resistance of several human eyes and found that the difference seldom exceeded 10 per cent. We do not think, therefore, that the milliammeter should be discredited.

PYROMETRIC SUPERVISION

Some ophthalmomologists, dissatisfied with the results obtained with the milliammeter as a control, looked for other means of control. Larson and Weve watched the reaction of the tissue in order to determine the proper dose of current. They increased the current until the sclera became parchment-like and a grayish-white coagulative spot appeared on the fundus. Coppez and, later, Meesmann, on the basis of research, suggested measuring the temperature of the eye on the site of origin of the heat by means of their special thermometric electrodes. It is a known feature of metals that whenever the soldered contact surface of two different metals is warmed up electric current is generated which is proportional to the difference in temperature between the contact surface and the room. If this electric current is measured by means of a galvanometer, the actual temperature of the contact surface can be computed in a simple manner, or the instrument itself can be calibrated in degrees of temperature. This so-called pyrometric principle has been employed by Coppez and Meesmann for measuring the temperature of the tissue during treatment. Coppez uses surface electrodes, while Meesmann uses pin-shaped ones.

In order to obtain the relation between electrical resistance and the temperature of the sclera and to check at the same time the advantage of the pyrometric method, we extended our investigations to such operations.

We used Meesmann pins only. We do not rely on the practical use of Coppez electrodes, for two reasons: 1. The use of the thermocouples on the surface makes it impossible to employ moistening, because, owing to its cooling effect, readings would be wrong. It has already been shown that the surface method is harmful if it is employed without moistening. 2. The thermo-electric pins, like all heat-measuring instruments, suffer from inertia; i. e., they react to the variations of temperature rather slowly. The retardation is the longer the larger their mass. This behavior of thermo-electric pins was pointed out by Coppez and Meesmann. Meesmann succeeded in designing his pins small enough to keep this inertia sufficiently low. Surface electrodes are much larger than pins; therefore their use as thermocouples does not appear to be justified.

We used the Meesmann pins for our tests. The process of coagulation, as could be seen from the curves for resistance, is the same as when the usual micropins are used. After connection of the diathermic current the resistance rapidly falls, remains constant on a lower level for a while and then rises again in consequence of drying up of the coagulated area, and at the end sparks occur.

The particulars of these tests are as follows:

With a current of 30 milliamperes the curve for resistance was stretched, and no slope appeared. The temperature of the sclera did not exceed 55 C. during an observation time of thirty-five seconds.

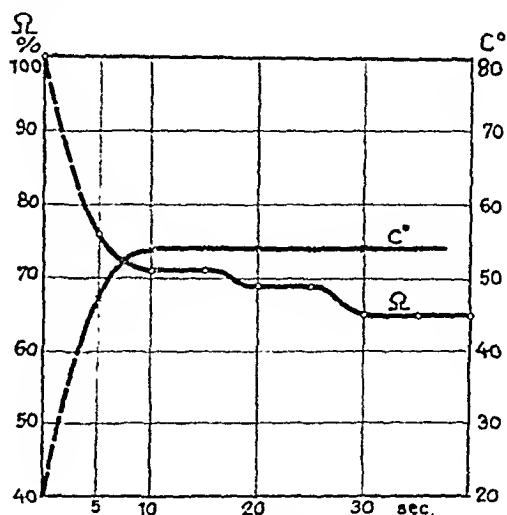


Fig. 6.—Variation of apparent temperature and resistance with time when a high frequency current of 30 milliamperes was used. The temperature was measured by means of pyrometric electrodes.

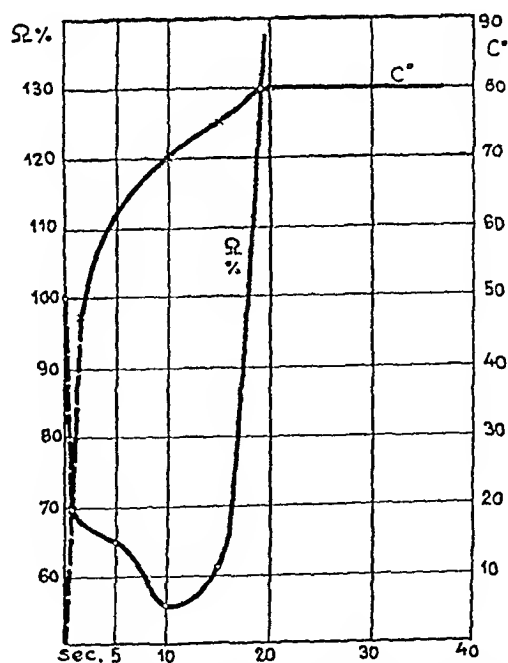


Fig. 7.—Variation of apparent temperature and resistance with time when a high frequency current of 38 milliamperes was used. The temperature was measured by means of pyrometric electrodes.

With a current of 40 milliamperes a temperature of 75 C. was indicated in four seconds. The resistance rose rapidly, and sparks occurred. Since a temperature of 75 C. does not explain the develop-

ment of sparks, retardation in the measurement of temperature must be supposed. After a few further seconds a rise of temperature could be read, but by this time exsiccation was already conspicuous.

Other tests displayed a further disadvantage of the pyrometric method. It may happen that, owing to the size of the pin or some condition of operation, the pin does not lie entirely in the sclera but part of the soldered surface is left in the air or the tip of the pin penetrates into the vitreous. Further, thermocouples have low electrical resistance and thus are not heated by the current itself but by the tissue only. Therefore, if partly heated they will not indicate the temperature of the sclera, but an average temperature between the temperature of the sclera and that of the air or the temperature of the vitreous part. This behavior was clearly demonstrated in the respective

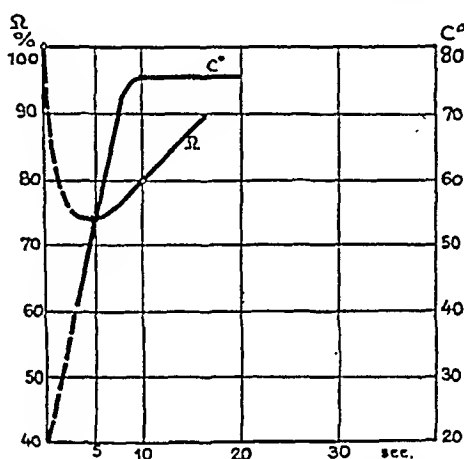


Fig. 8.—Variation of apparent temperature and resistance with time when a high frequency current of 40 milliamperes was used. The temperature was measured by means of pyrometric electrodes.

curves for resistance and for temperature obtained during these tests. This disadvantage is, of course, eliminated if the soldered contact surface lies entirely in the sclera.

One learns from these tests that while the pyrometric method gives valuable data about the process of coagulation it does not satisfy the requirements of a proper control of the dosage if used unaccompanied by some other measurement. If used alone it may easily deceive the operator about the true state of coagulation, since as we could see sparks and carbonization occurred before the rise in temperature was indicated by it. Its use would be greatly facilitated if the size of the pins could be diminished to the size of pins used in micropuncture.

Contrary to the pyrometric method, the measurement of the resistance appeared to be, as might be expected from the theory, instantaneous, inertialess and reliable in every respect. The horizontal part

of the curve insures sufficient time for the surgeon to watch the coagulation, and the current can be disconnected before the drying up of the sclera, i. e., before the rise of the curve for resistance begins. After some experience this method of measuring resistance will be found to be easy, especially if the apparatus and diathermic current remain unchanged through a series of operations.

COMMENT

The success of the operation for healing retinal detachment depends chiefly on the diathermic coagulation, since the irritation producing adhesive choroiditis is the effect of coagulation. It is important that the irritation be kept between certain limits, because violent as well as too weak irritation may do harm. As far as we could see, there are several important factors influencing coagulation. The individual factor of the eye under treatment may be disregarded, as a careful choice of method and dosage insures the reproduction of such operative conditions as have been proved by former experiences of the surgeon. Four factors should be kept in mind:

- (1) the type and shape of the electrodes;
- (2) the diathermic-apparatus, i. e., the generator of the high frequency current;
- (3) the frequency of the current, and
- (4) the rate of the current.

1. *Electrodes*.—The heat generated depends on the amount of the surface of the electrodes touched by the tissue and on the piercing depth of the pins. The shape and size of the electrodes should be the same for all operations. If micropuncture is employed the pins should be as thin as possible, and the piercing depth should be the same for all the operations. To insure the proper depth of piercing the best practice is to provide the pin with an isolated stopping plate. If surface coagulation is applied the electrode and the pressure of the hand should be the same for all the operations. On the application of surface coagulation one should never omit moistening the sclera, in order to avoid rapid drying up and irregular coagulation.

2. *Diathermic Apparatus*.—The surgeon should use only that type of diathermic apparatus the operation of which is thoroughly known to him. It will be found useful to calibrate the apparatus for power of coagulation by the method employed in the tests already described. Although the electric power required for the operation is rather low, the use of a high power apparatus is recommended, in order to avoid fluctuations of voltage when the load is varying.

3. *Frequency of Current*.—High frequency current seems to be more suitable for the production of coagulation than low frequency

current. It must be noticed, however, that should the former be employed the proper adjustment of the dosage will be more difficult. This is obvious from the following considerations: The milliammeter of the apparatus shows in most cases the total current afforded by the apparatus. Capacitive current may leak partly from the leading-out wires of the apparatus and partly from the eye itself. This current does not take part in the heating of the eye, and the larger is the portion it forms the higher the frequency of the total current. The fault caused by capacitive current is essentially diminished, and it can be diminished still more if one calibrates the apparatus according to the suggested method of the measurement of resistance.

4. *Rate of Current.*—It is seen from the results of the test that the duration of the horizontal part of the curve for resistance can be influenced by adjusting the rate of the current. If the current is low, coagulation will be too slow, and the required time of operation will be inconveniently long. If it is high, carbonization may occur so rapidly that one may easily miss the proper time to disconnect the current and may harm the eye. We found it best to adjust the current to a value involving a coagulation time of from four to six seconds. The duration of the operation has, further, an influence on the process of coagulation. The extension and depth of the coagulating area increase in proportion with the duration of the procedure, which is, however, advantageous to a certain limit only.

We found that the optimal current taken from the surgical diathermic apparatus described amounted to about 30 milliamperes for micropuncture and from 50 to 70 milliamperes for surface coagulation. It is possible that this value would differ somewhat if another apparatus were used; e. g., 50 milliamperes read on an apparatus may have an effect equal to that of a current of 60 milliamperes of another apparatus. It would be appropriate if some standardizing station would calibrate all such apparatus according to equal effects, using standard electrodes and standard substitutes for the living eye.

CONCLUSIONS

The chief purpose of our experiments was to find out whether the dose in diathermic irritation can be specified and whether the specified dose can be accurately adjusted during the operation and, further, to find available means to do so.

Both questions may safely be answered in the affirmative. The result of our experiment furnished the proof that the dose can be specified and adjusted on the basis of the milliammeter reading when it is supposed that the apparatus has been calibrated for the coagulating capacity with the same arrangement and same electrodes used for car-

rying out the operation. Larsson went still further and tried to abandon the use of the milliammeter. We do not agree with him. It is admitted that the current can be adjusted for all similar operations, and a milliammeter reading seems to be superfluous. It was found, however, as mentioned in this paper, that the reading gives an indication of the variations of current and of the drying up of the coagulating spot.

It must be stated, however, that the milliammeter reading alone is not to be relied on solely. Biologic control of the process by ophthalmoscopy is required during operation. While the proper dosage is adjusted by electrical methods the results are checked by ophthalmoscopy to determine whether the desired effect has taken place on the desired spot.

From the results of the research described in this paper it may be stated that the dosage of diathermic coagulation can be checked and the dangers indicated by Vogt can be easily avoided.

OCULAR SIGNS OF THROMBOSIS OF THE INTRACRANIAL VENOUS SINUSES

FRANK B. WALSH, M.D.

BALTIMORE

While the ocular signs of thrombosis of the cavernous and lateral sinuses are well known, there is not complete agreement regarding the mechanism producing them. The ocular changes of thrombosis of the superior longitudinal sinus are less thoroughly understood. Some of the conditions hitherto diagnosed as serous meningitis, pseudotumor of the brain or chronic arachnoiditis may be due to thrombosis of the venous sinuses in the presence of an abnormal venous sinus pattern. In this paper the ocular signs of thrombosis of these dural sinuses are described, and an effort is made to correlate some of these with the anatomicopathologic findings in septic thrombophlebitis of the cavernous and lateral sinuses.

In approaching the subject I shall first review the relevant pathologic and anatomic features.

PATHOLOGIC PICTURE

Thrombi may be aseptic (primary, marantic or autochthonous) or septic.

The aseptic form of thrombosis occurs usually in the superior longitudinal sinus, less frequently in the lateral sinus and least frequently in the cavernous sinus (Uhthoff).¹ Septic thrombosis occurs most often in the lateral sinus. The septic form is more common than the aseptic. Macewen² in 1893 differentiated these forms as follows: Aseptic thrombosis characteristically (1) occurs in the nonpaired sinuses, (2) is rarely associated with purulent infection, (3) shows a tendency to organization or resorption, (4) is rarely complicated by meningitis and (5) in one-half the cases is followed by extravasation into the brain and a tendency to softening. Septic thrombosis is characterized by (1) occurrence in the paired sinuses, (2) frequency of purulent infection, meningitis and cerebral abscess, (3) a tendency to

From the Wilmer Ophthalmological Institute, the Johns Hopkins Hospital.

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1. Uhthoff, W., in von Graefe, A., and Saemisch, E.: *Handbuch der gesamten Augenheilkunde*, Berlin, Julius Springer, 1911, vol. 11, p. 697.

2. Macewen, W.: *Pyogenic Infective Diseases of Brain and Spinal Cord*, Glasgow, J. Maclehose and Sons, 1893, p. 226.

purulent degeneration of the thrombus and (4) rare occurrence of extravasations into the cerebrum and cerebellum.

Thrombosis and phlebitis are usually regarded as reciprocal processes. The relative importance of slowing of the blood stream and infection of the vein as causative factors was a subject of debate until Welch showed that infection of the vein precedes the formation of the thrombus in most cases. Aseptic thrombosis may occur as a result of injury, but it may also occur as a result of changes in the blood itself or from dehydrating diseases.

Ordinarily thrombi grow in the direction of flow of the blood stream, but they may develop in the opposite direction. Such retrograde development is frequent in the intracranial venous sinuses, where it may be accounted for by the absence of valves in many of the venous channels as well as by the plentiful collateral circulation.

Thrombi may cause vascular obstruction. Such obstruction accounts for certain of the ocular signs and may result in damage to the brain through infarction, necrosis and hemorrhage. Septic thrombi result in bacteremia and septicemia and through direct extension may give rise to abscess of the brain and meningitis.

Septic thrombophlebitis is constantly accompanied by edema of the surrounding tissue. This edema is due, for the most part, to lymphangitis (Boyd³).

ANATOMIC PICTURE

The cavernous sinus is situated at the side of the body of the sphenoid bone. It extends from the superior orbital fissure to the tip of the petrous portion of the temporal bone, a distance of slightly more than 2 cm. The sphenoid sinus lies medially, and the middle cranial fossa with the temporal lobe lies lateral to it, while the hypophysis lies medial to and above it. The sinus is made up of many compartments, which become fewer as age advances. It contains the carotid artery and the sixth nerve, which lies lateral to the artery. The third, fourth and ophthalmic (sometimes the mandibular as well) divisions of the fifth nerve lie in its lateral wall in that order from above downward. Anteriorly it receives the sphenoparietal sinus, the superior ophthalmic vein, which in turn receives the anterior and posterior ethmoid veins and veins from the frontal diploe, and the inferior ophthalmic vein, which drains the outer portion of the floor of the orbit. The inferior ophthalmic vein communicates with the facial veins and with the veins of the pterygoid plexus. The middle cerebral vein and veins from the hypophysis and from the sphenoid sinus empty directly into the cavernous sinus. Through communications with the precentral and

3. Boyd, W.: *Surgical Pathology*, Philadelphia, W. B. Saunders Company, 1925, p. 141.

postcentral veins, which empty into the superior longitudinal sinus, the middle cerebral vein establishes communication between the superior longitudinal and the cavernous sinus. Through emissary veins the cavernous sinus communicates with the pterygoid plexus and by the carotid plexus with the internal jugular vein. Posteriorly, it gives off the superior and inferior petrosal sinuses. The superior petrosal sinus runs in the attached margin of the tentorium cerebelli above the trigeminal nerve and empties into the lateral sinus. The inferior petrosal

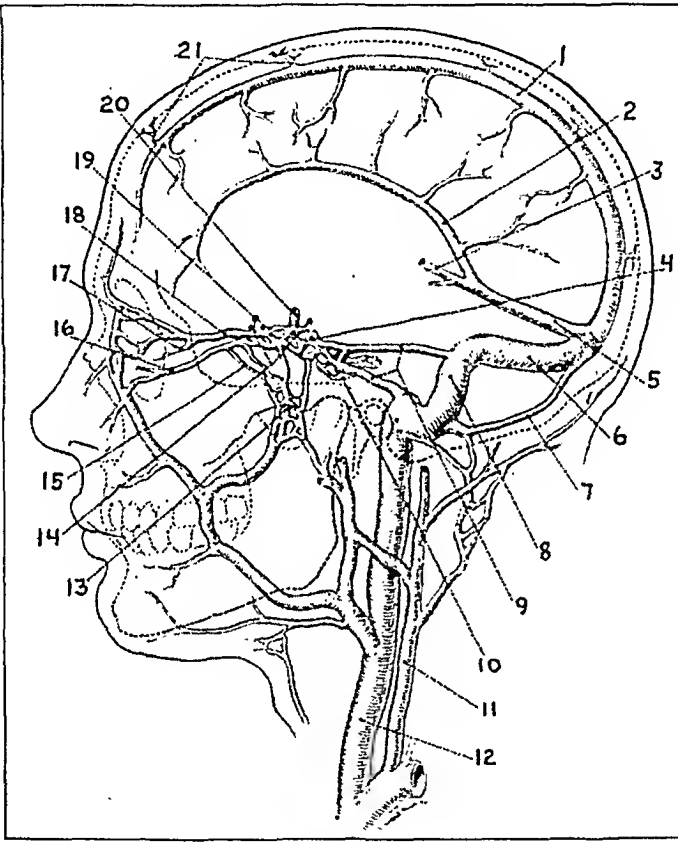


Fig. 1.—Drawing prepared by Dr. Frank R. Ford showing 1, superior longitudinal sinus; 2, inferior longitudinal sinus; 3, vein of Galen; 4, cavernous sinus; 5, straight sinus; 6, lateral sinus; 7, occipital sinus; 8, sigmoid sinus; 9, superior and inferior petrosal sinuses; 10, basilar sinus; 11, external jugular vein; 12, internal jugular vein; 13, pterygoid plexus; 14, intercavernous sinuses; 15, communication between inferior ophthalmic vein and pterygoid plexus; 16, inferior ophthalmic vein; 17, central vein of retina; 18, superior ophthalmic vein; 19, sphenoparietal sinus; 20, middle cerebral vein, and 21, cerebral veins.

sinus, shorter but larger than the superior, passes with the sixth nerve below the petrosphenoid ligament and enters the internal jugular vein. Each cavernous sinus communicates with its fellow of the opposite side through the anterior and posterior intercavernous sinuses.

The superior longitudinal sinus commences at the foramen caecum and extends posteriorly in the attached margin of the falx cerebri to the internal occipital protuberance, where it usually turns to the right as the right lateral sinus. The superior longitudinal sinus receives four main tributaries: the frontal, precentral, postcentral and occipital veins. It drains the greater part of the external and the superior mesial surface of the cerebral cortex.

The inferior longitudinal sinus occupies the free margin of the falx cerebri. It is continuous with the straight sinus, which usually turns to the left and is continued as the left lateral sinus. The inferior longitudinal sinus receives blood from the mesial surface of the cerebral cortex.

The great cerebral vein (vein of Galen) drains into the straight sinus. It drains the greater part of the white matter and the basal nuclei.

The lateral sinus on each side commences at the internal occipital protuberance and runs laterally in the border of the tentorium to the base of the petrous bone, where it leaves the tentorium and passes down through the jugular foramen as the internal jugular vein.

The venous sinuses near the torcular show many variations. Five types may be described: (1) the common pool, which is pictured in most textbooks, (2) the plexiform, in which there may be an adequate cross-circulation between the two lateral sinuses, (3) the ipsilateral, in which a separate cross-channel from one lateral sinus to the other may be present and thus provide cross-circulation, (4) the unilateral, in which the straight and superior longitudinal sinuses empty into the lateral sinus of one side, and (5) the occipital, in which the occipital sinus may be of sufficient size to transport a large volume of blood.

SEPTIC THROMBOPHLEBITIS OF THE CAVERNOUS AND LATERAL SINUSES

The following observations, except when otherwise stated, are based on clinical and autopsy studies of patients in the Johns Hopkins Hospital. The histories and autopsy records are summarized at the end of the section.

Thrombophlebitis of the cavernous sinus was observed at autopsy in all these cases. The following were the salient clinical findings.

Exophthalmos and Chemosis.—Exophthalmos was usually bilateral but might be unilateral (case 6). In a case reported by Keegan and Ash⁴ it was absent. The amount of chemosis appeared to parallel the amount of exophthalmos.

4. Keegan, J. J., and Ash, W. E.: Bilateral Cavernous Sinus Thromboses Without Involvement of the Ophthalmic Vein: Report of Cases, Arch. Ophth. **12**: 72 (July) 1934.

Exophthalmos appeared to be mainly dependent on infection of the orbital tissues. Such a finding was present in all the cases reported here. Uththoff expressed the belief that if infection does not proceed forward from the cavernous sinus, resulting in infection of the orbit, exophthalmos does not appear. Vascular obstruction, however, must play a part, since exophthalmos occurs in nonseptic thrombosis, and otherwise partial recession of a proptosed eye in a fatal case, such as was noted in case 6, would not occur.

The degree of exophthalmos was variable. It was less marked when the cavernous sinus was involved through a retrograde extension from the lateral sinus than when it originated from an anterior infection. Eagleton⁵ referred to these cases as instances of a "chronic compensatory" type and remarked that in them the exophthalmos might be transitory.

Macewen stated that coincident with the involvement of the second eye there may be apparent improvement of the condition of the first eye.

Edema of the Lids.—This was a striking feature in cases of fulminating thrombosis, in which the thrombosis usually arises from an anterior infection. The upper lid was affected to a greater degree than the lower lid. The swelling of the lid may interfere mechanically with motion of the lid, so that paralysis of the levator may pass unnoticed. The lids are freely supplied with lymphatics. It appears that infection rather than vascular obstruction accounts for swelling of the lids. Faulkner⁶ has shown that swelling of the lower lids is not pathognomonic of thrombosis of the cavernous sinus and may be due to infection of the antrum or the ethmoid sinus.

Paralysis of the Muscle of the Eye.—In cases of fulminating thrombosis there was early paralysis of the extra-ocular muscles on the affected side. Ptosis came on early. Behr,⁷ among others, noted that it might be purely mechanical and that external ophthalmoplegia might be simulated by edema of the lids. He expressed the opinion that ophthalmoplegia is due to a lesion of the nerves in the cavernous sinus, caused either by pressure or by inflammation. It seemed impossible to establish either infection or pressure in the orbit or in the sinus as a single cause of muscle palsies.

5. Eagleton, Wells P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses: A Clinical Study of Blood Stream Infection, New York, The Macmillan Company, 1926.

6. Faulkner, E. Ross: Surg., Gynec. & Obst. 52:474, 1931.

7. Behr, Carl, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 6, p. 229.

Internal ophthalmoplegia was rarely encountered at the first examination and was noted only once in this group of cases (case 1). It is usually described as a late symptom.

Parsons⁸ has stated that paralysis of the external rectus muscle of the second eye is the first sign of its involvement. Such paralysis is probably to be explained either by basilar meningitis or by increased intracranial pressure.

In thrombosis of the lateral sinus the onset of paresis of the sixth nerve frequently signifies an extension forward of the process via the inferior petrosal sinus, which is the usual route by which the thrombotic process extends from the lateral to the cavernous sinus (cases 2 and 5). The involvement of the sixth nerve may be accompanied with pain in the face and behind the eye on the same side, due to involvement of the fifth nerve.

Transient improvement of external ophthalmoplegia was an unusual observation in case 6, in which there was also partial recession of the proptosed eye. The causal mechanism of this occurrence was not obvious. Conjugate deviation is unusual and was not observed in the cases reported here.

Some of these patients were so ill that cooperation could not be obtained in estimating the presence or absence of muscle palsies. In one such instance irrigation of the aural canals was a useful maneuver (case 5) when appropriate nystagmus indicated the absence of complete paralysis.

Nystagmus.—Nystagmus is infrequent and was not noted in the cases reported here.

Corneal Changes.—Increased corneal sensitivity was observed (case 5), but it soon gave way to anesthesia of the cornea. Cloudiness and necrosis of the cornea may be observed when there is exposure of the cornea, but frequently no change is seen.

Ophthalmoscopic Signs.—The ophthalmoscopic signs of thrombosis of the cavernous sinus are of slight importance in establishing the diagnosis. These signs are engorgement of the veins and low grade papilledema. The fundi, however, may remain normal throughout the course of the disease (Parsons). This appeared to be so in case 5 reported here. However, the eyes were not examined on the day preceding death and at autopsy showed low grade papilledema. Benedict⁹ observed these signs in all his cases but remarked on their late appear-

8. Parsons, J. Herbert: *The Pathology of the Eye*, New York, G. P. Putnam's Sons, 1908, vol. 4, p. 1226.

9. Benedict, W. L.: *Surg., Gynec. & Obst.* 52:262, 1931.

ance. Microscopic examination of the eyes in the cases reported here supported the view that papilledema is present in every case.

Generalized retinal edema may be apparent. This was observed in one case in the group reported here (case 4). Sections of the eye in this case did not differ from those in the remaining cases, in all of which there was retinal edema.

Pulsation of the retinal arteries was observed in one patient (case 1). This was due to pressure from swollen lids and orbital structures.

Hemorrhages are infrequent, and consequently, it may be inferred that thrombosis of the central vein is infrequent. Hemorrhages were not observed in the cases reported here.

Abscess formation in the eye apparently does not occur, although examination of the choroid always shows many cells, mostly mononuclears and lymphocytes and a few polymorphonuclears and leukocytes.

Visual Acuity.—It is usually impossible to chart the vision with any accuracy. It is frequently greatly reduced. In the cases reported here accurate records of visual acuity were not available.

Visual Fields.—There is nothing characteristic about the visual fields. In the cases reported here records of the visual fields were not attempted.

REPORT OF CASES

The summaries of the clinical histories and autopsy notes contain only the essential facts.

CASE 1.—E. C., a Negro woman aged 27, acquired a sore throat ten days before her admission to the Johns Hopkins Hospital. Three days after her throat became sore the left side of her face swelled, and a few days later the right side of her face swelled. The swelling on the left side receded. She had been extremely ill for several days prior to admission.

The temperature was 105 F., the pulse rate 142 and the respiratory rate 40. The patient was irrational. Her neck was stiff, and there was a positive Kernig sign on the right. The right side of her face was greatly swollen. She was unable to open her mouth. The lids of the right eye were edematous and greatly swollen, and folds of edematous conjunctiva protruded between them. The left upper lid was swollen. There was total ophthalmoplegia on the right and paralysis of all the extra-ocular muscles on the left, except the internal rectus muscle, which was partially paralyzed. The right eye was definitely proptosed, and the left eye possibly was slightly proptosed. There was venous congestion of the retina, more apparent on the right. The tonsils were reddened and swollen, but there was no evidence of peritonsillar or retropharyngeal abscess. The glands on the right side of the neck were swollen and tender.

The white cell count of the blood was 30,000. The spinal fluid contained 4,000 cells per cubic millimeter, mostly polymorphonuclears. Organisms could not be cultured from the spinal fluid. Blood cultures were negative.

The patient died on the second day after admission.

Autopsy.—There was abscess formation in each orbit. A purulent exudate extended over the surface of the brain, particularly over the base. There was a hemorrhage over the right frontal lobe. The cavernous sinuses were filled with

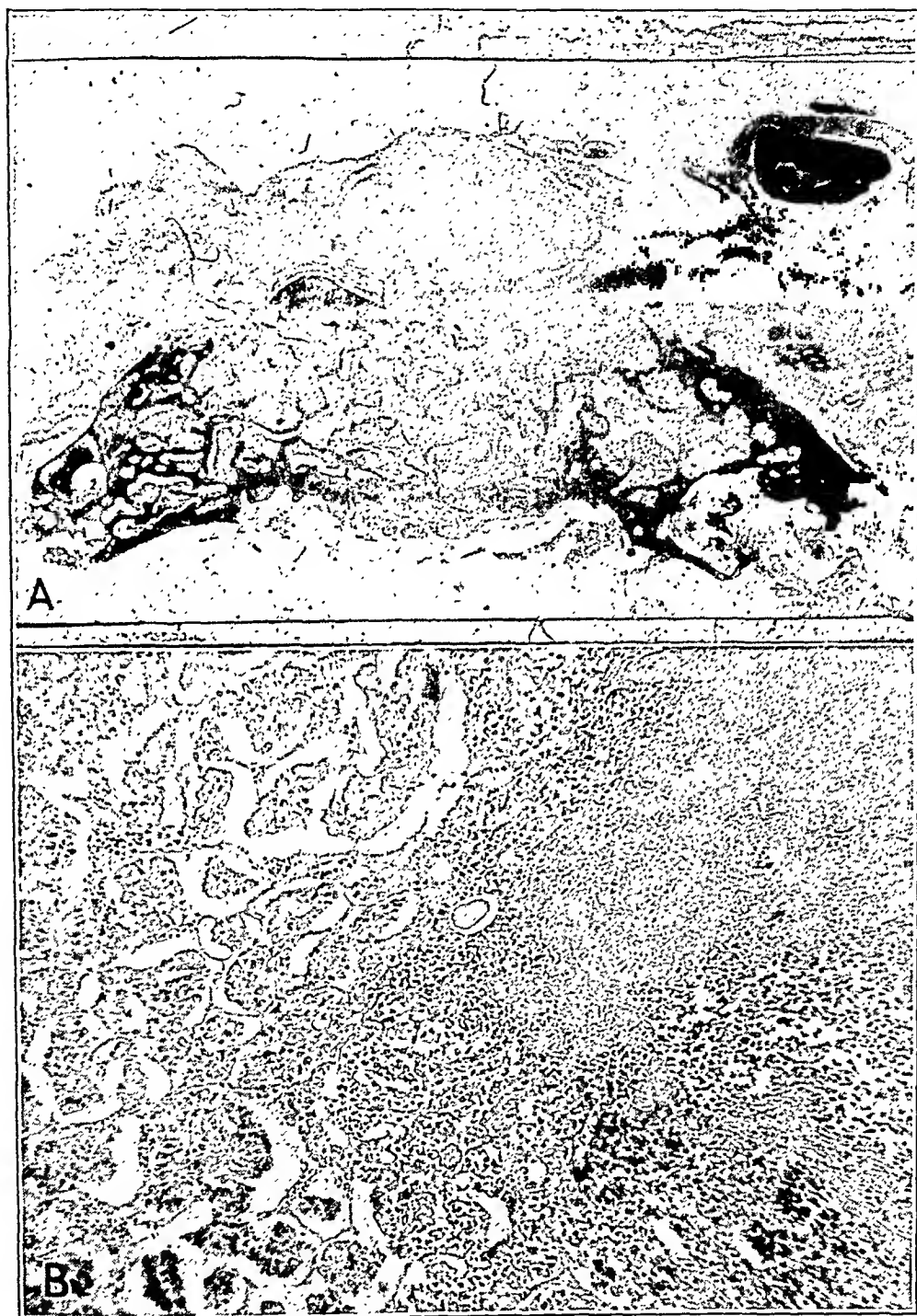


Fig. 2.—Sections from the hypophysis showing (A) general involvement and (B) necrosis and early abscess formation.

purulent material. The hypophysis contained an area of necrosis about the center of the anterior lobe, in which bacteria were present. The area was surrounded by inflammatory cells, many of which were polymorphonuclears. Pus was observed

extending along the floor of the sella turcica. The lateral and superior longitudinal sinuses were clear.

Bacterial stains showed gram-positive cocci in the hypophysis, cavernous sinuses, sphenoid sinuses and meninges.

CASE 2.—C. M., a Negro boy 7 years old, had a cold two weeks before admission to the hospital. A week after the onset of the cold, his left ear became painful and he complained of pain in the left side of his face. A paracentesis of the left ear drum failed to relieve the pain.

On the day of admission the temperature was 104 F., the pulse rate 124 and the respiratory rate 28. The head was turned to the right. There was tenderness over the tip of the left mastoid process. The left external rectus muscle was incompletely paralyzed. There were no other ocular signs. Other examinations, including the usual laboratory tests, failed to add anything to the clinical picture.

On the day after admission a simple mastoidectomy was performed on the left side. The mastoid antrum was opened and was found to contain pus, but there did not appear to be any extension of the process. There was no improvement in the patient's general condition, and consequently the wound was opened and the lateral sinus was examined. It did not appear to be thrombosed. Six days after admission the upper lid of the left eye became swollen and the neck became stiff. Within a day there was complete ophthalmoplegia on the left, and after another day the right eye was similarly affected. There was no ophthalmoscopic evidence of vascular obstruction until two days before his death, when there was slight venous overfilling.

Death occurred nine days after the boy's admission to the hospital.

Autopsy.—There was abscess formation in both orbits and purulent thrombophlebitis of both cavernous sinuses and of the left petrosal sinus. There was a subdural abscess on the left side and localized pachymeningitis and leptomeningitis. Layers of infected blood clots were present in the left middle cranial fossa, in the sphenoid sinus on the left and in the superior longitudinal sinus. The lateral sinus was patent on both sides. The torcular herophili was patent. An abscess 1 cm. in diameter was continuous with the left cavernous sinus. The nerves in the left sinus were invaded by inflammatory cells. One section of the carotid artery showed pus in its lumen. There was necrosis of a portion of the anterior lobe of the hypophysis.

The origin of this widespread thrombophlebitis is not clear. The fact that the clinical symptoms suggested involvement of the inferior petrosal sinus on the left and that pus was found in the mastoid antrum makes it appear probable that it originated in the lateral sinus.

CASE 3.—J. J., a Negro aged 41, was admitted to the Johns Hopkins Hospital complaining of a swollen left ear. He had had a chancre twenty years before and also a discharging left ear. Ten days before admission he caught cold, and on the following day there was an increased amount of discharge from the left ear, which had discharged irregularly for many years. Two days before admission the lids of the left eye swelled and he suffered from headache and chills.

On examination his temperature was found to be 101.4 F. and the pulse rate 100. There was marked boggy edema of the lids of the left eye. There were inability to move the left eye up or down and almost complete loss of lateral movement; the right eye moved normally. The pupils reacted normally to light. There was slight engorgement of the veins of the left eye. There were palpably

enlarged, tender glands on the left side of the neck. There was a thick, creamy discharge from the left ear, but no tenderness of the mastoid.

General examination showed an infection of the upper respiratory tract. Laboratory and roentgen examinations failed to add to the clinical picture.

Two days later there was definite involvement of the right eye. Both eyes became slightly proptosed and showed external ophthalmoplegia. There was slight clouding of the cornea. Ophthalmoscopic examination revealed slight evidence of venous engorgement and possible slight papilledema.

Death from septicemia occurred four days after the patient's admission.

Autopsy.—Both orbits were invaded by inflammatory cells, and the right cavernous sinus was thrombosed, with the thrombus extending into the left cavernous sinus. At the base of the sphenoid on the left the dura was separated



Fig. 3.—Areas of necrosis in the anterior lobe of the hypophysis.

from the bone by a soft bulging mass. The hypophysis showed microscopic evidence of necrosis of the anterior lobe, and although there was no gross evidence of meningitis, microscopic examination showed the presence of an inflammatory exudate in the meninges. Some of the veins surrounding the left carotid artery were thrombosed. The left mastoid process was eburnated from long-standing infection. There was an inflammatory membrane in the pharynx. Gram-positive cocci were found throughout the infected areas. This infection probably originated in the throat and spread to the cavernous sinus via the carotid venous plexus.

CASE 4.—C. S., a Negro aged 40, suffered from toothache for ten days before admission to the hospital. This resulted in swelling of the left side of the face and inability to open his mouth. Two days before admission the lids of the left eye swelled, and a day later those of the right eye also. He had felt feverish for several days prior to admission.

Examination showed bilateral swelling of the lids, which could not be opened voluntarily. There were bilateral paralysis of the external rectus muscle and limitation of the movements obtained through the other muscles. The pupils reacted promptly to light. The corneas appeared normal, but there was increased sensitivity on the right. Ophthalmoscopic examination showed slight edema of the whole retina in each eye. There was a low grade papilledema. The left side of the face was greatly swollen. Difficulty was experienced in examining the mouth. Several teeth in the left side of the upper jaw were bathed in pus. General examination and the usual laboratory tests did not add anything to the clinical picture.

The swelling of the lids rapidly increased. The external ophthalmoplegia became complete. There was no appreciable change in the ophthalmoscopic appearance of the fundi.

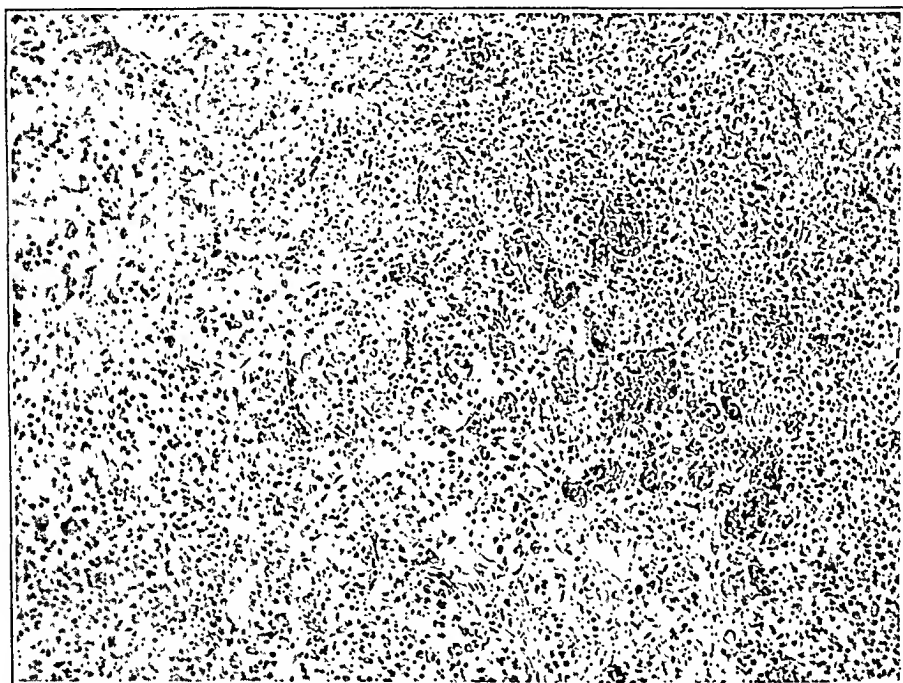


Fig. 4.—Areas of necrosis in the hypophysis.

Autopsy.—There were infected thrombi in the veins in each orbit and orbital infection. There were bilateral thrombosis of the cavernous sinus, acute osteomyelitis of the left sphenoid and left temporal bone and localized pachymeningitis and leptomeningitis on the left, with extension to the left temporal lobe. The dura on either side of the body of the sphenoid on its posterior surface was reddened, necrotic and overlying purulent material. There were multiple areas of necrosis in the hypophysis. Inflammatory cells were seen in the gasserian ganglion.

An interesting feature was the absence of meningitis. The infection appeared to have originated in the jaw and spread to the left orbit through the veins of the face and to have more directly infected the cavernous sinus through the pterygoid plexus. Infection of the cavernous sinus may have been via the sphenoid sinus.

CASE 5.—M. L., a white girl aged 2 years, was admitted to the hospital with a diagnosis of acute mastoiditis on the right, and questionable mastoiditis on the left. A simple mastoid operation was performed on the right, but it was not followed by improvement in the general condition. Three days later operation was performed on the other side. The condition became critical, and on the day after the last operation a positive Queckenstedt reaction was obtained on the left. The left internal jugular vein was tied off. One day later there was swelling of the lids of the left eye and of the left side of the face. At that time there were no extra-ocular palsies. The eyegrounds appeared to be normal except for a slight and questionable edema around the right disk.

Two days later there were great swelling of the lids of the left eye and slight swelling of those of the right eye. The left eye was slightly proptosed. There

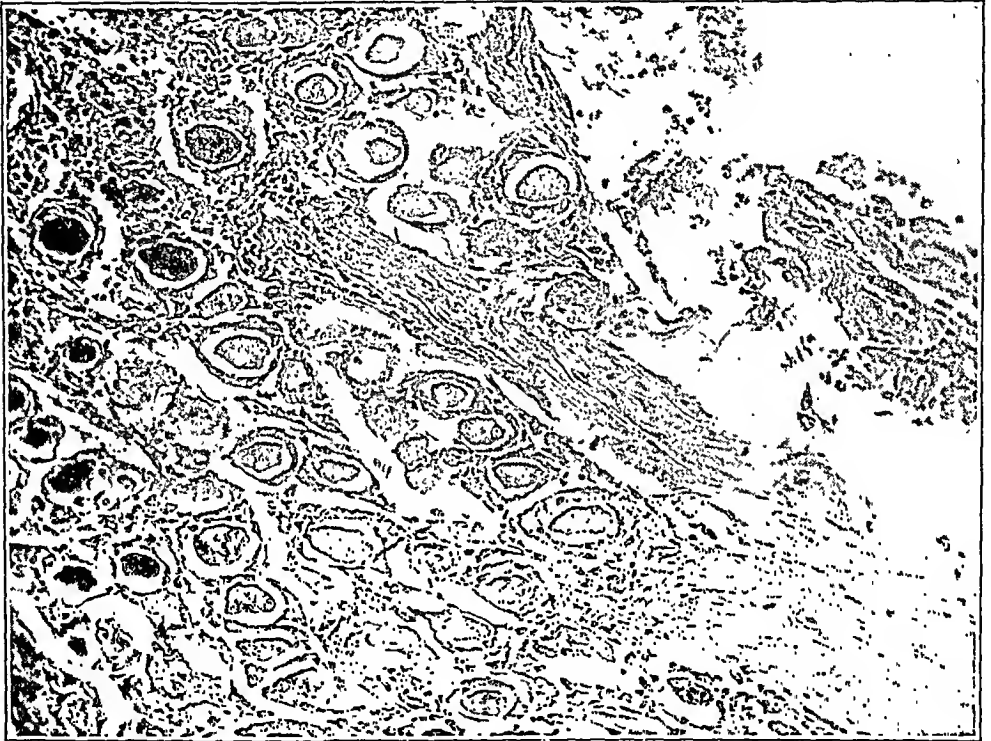


Fig. 5.—Inflammatory cells in the gasserian ganglion.

was no change in the appearance of the fundi. The pupils were semidilated and reacted actively to light. There was apparent bilateral external ophthalmoplegia.

Irrigation of the aural canals with water, either hot or cold, produced appropriate nystagmus. These responses disappeared after twenty-four hours.

Death occurred nine days after the patient's admission to the hospital.

Autopsy.—There were abscesses in each orbit and a purulent exudate beneath the periosteum in the region of the frontoparietal suture on the left side. Either the middle meningeal vein or the artery was thrombosed. Both cavernous sinuses contained purulent material and were bathed in pus. Both lateral sinuses contained infected clots. The thrombus was small and gray in the right lateral sinus and a fresher red-gray in the left lateral sinus. The superior longitudinal sinuses and the straight sinuses were patent. An abnormality was noted in that a communicating branch between the right superior petrosal sinus and the middle

meningeal vein was present. The dura was loosened from the skull over the petrous portion of the left temporal bone and over the left sphenoid at the base. There were an abscess of the hypophysis and necrosis of the sphenoid.

In this case there was a bilateral infection of the lateral sinuses, which appeared to have spread forward to the cavernous sinuses and thence to the orbits.

CASE 6.—L. L., a white boy aged 15, was admitted to the hospital on March 18, 1936. He complained of pain in the right ear, discharge from that ear, frontal headache, recurrent chills and pains in the joints. He had had a discharging ear for over a year prior to his admission. One week before admission he had received tetanus antitoxin for an accidental burn. Ten days before admission he went swimming. This resulted in increased discharge from the ear. After a few days the ear became painful. He visited the dispensary on several occasions. There was no evidence of involvement of the mastoid. On March 18 he complained of severe frontal headache and of chills on the previous night.

The temperature was 103.4 F. and the pulse rate 106. There were tenderness of the right wrist and elbow, redness and swelling about the site of injection of the tetanus antitoxin and swelling of the glands in the right axilla. There was a copious discharge from the right ear. The eyes were normal on external examination. The vision was not tested. There was definite blurring of both disk margins, more marked on the right. The leukocyte count was 14,400. He was thought to be suffering from serum sickness, but because of the discharging ear and ophthalmoscopic signs he was admitted to the wards.

On March 20 a radical mastoidectomy was performed on the right. The lateral sinus was thrombosed. The sinus was opened, the clot evacuated and the sinus packed. The internal jugular vein was ligated. On the following day and for several days the patient felt better, but there was a continuation of the septic fever. On the sixth postoperative day he complained of a sense of fulness about his eyes. There were definite edema of the upper lid of the right eye and inability to elevate it normally, which appeared to be due to involvement of the nerves. There was a proptosis of 4 mm. The left eye appeared to be normal. The vision in each eye was acute. Ophthalmoscopic examination showed a bilateral papilledema, more marked in the right eye but not of sufficient amount to measure accurately. There was slight chemosis. On the seventh postoperative day there were complete paralysis of the external rectus muscle on the right side and inability to move the eye fully in any direction. On the following day the right eye could be moved in any direction almost normally and could be abducted fully, but the ptosis remained unchanged. There was at that time no change in the appearance of the fundi.

The packs were removed, and a quantity of foul-smelling pus was allowed to escape.

On the ninth postoperative day the patient became irrational, and swelling on the left side of the scalp was observed. On the next day there were a return of the paralysis of the right external rectus muscle and an almost complete paralysis of the other extra-ocular muscles of the right eye, and paralysis of the left external rectus muscle appeared. The amount of proptosis of the right eye was decreased definitely. There was no change in the appearance of the fundi.

Autopsy.—There was mastoiditis on the right, with erosion of the temporal bone. The right orbit showed abscess formation. The cavernous sinuses were filled with pus. The right transverse, inferior and superior petrosal sinuses were thrombosed. There was an abscess of the left temporal lobe. Meningitis was present.

COMMENT

Changes Observed at Autopsy.—In the cases reported here the changes observed at autopsy may be summarized as follows:

Orbits: In every instance infection of the orbital tissues was present. Infiltration of the muscles with inflammatory cells was frequent, as was thrombosis of the orbital veins.

Cornea: Clouding and necrosis of the cornea were observed clinically, but only posterior sections of the eyes were obtained for study.

Retina: Edema of the retina was present in all cases. It is notable that it was not more marked in the instance in which it was observed ophthalmoscopically than in the cases in which it was not observed. It probably appears late in most cases.

Choroid: Inflammatory cells were present in the choroid in all cases. These were mostly mononuclears and lymphocytes, with only a few polymorphonuclear leukocytes. In no case was there abscess formation in the eye.

Optic Disks: Low grade papilledema was present in all cases. Thrombosis of the central retinal vein was not observed.

Nerves: Inflammatory changes were noted in the nerves in one instance and in the gasserian ganglion in another.

Cavernous Sinuses: Owing to the tendency for septic thrombi to disintegrate and liquefy early, microscopic examination may be essential in the pathologic diagnosis of thrombophlebitis of the cavernous sinus.

Hypophysis: This structure was affected in five of six cases. The stalk of the hypophysis appeared normal in the one instance in which it was examined. Necrosis with or without inflammatory cells is the usual lesion. This probably arises through infarction, which, however, would be difficult to demonstrate, as it would necessitate cutting serial sections.

Involvement of the hypophysis has not been stressed in previous reports on septic thrombophlebitis of the cavernous sinus. In those instances in which a clinical diagnosis of cavernous sinus thrombosis is made it is usually only of academic interest, but in cases in which recovery takes place signs of hypophysial dysfunction may occur.

It is probable that thrombosis of the cavernous sinus with recovery is relatively frequent, for the diagnosis is made only when ocular signs are present, and it is fairly frequently noted unexpectedly at autopsy.

I wish to present the essential facts regarding a case in which thrombosis of the lateral sinus ultimately resulted in Fröhlich's syndrome and extreme loss of vision.

K. C. P., a white boy of 11 years, entered the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital on Aug. 2, 1933. He complained of blind-

ness of the right eye and marked loss of vision of the left eye. The family history was irrelevant. He had had abscesses in the ears as a small child.

Present Illness.—The patient's mother dated the onset of his present illness from the occurrence of mastoiditis on the right side two years before the date of the admission. The symptoms of mastoiditis were present for two weeks before operation was performed. Operation was followed by a stormy course, and ten days later the jugular vein was ligated. Tonic deviation of the eyes to the right was noted after this operation. Five days later polyuria and polydipsia developed, which persisted for six or eight weeks. His recovery was further complicated by phlebitis of the right leg and an infection of the hip joint. He gradually lost vision in both eyes. After the acute signs had subsided, there was no light perception in the right eye and the vision in the left eye was 3/200. The optic disks were reported to be swollen and to have connective tissue about them.

Examination.—The boy presented the characteristic signs of Fröhlich's syndrome. General and neurologic examinations gave negative results. Otologic examination showed the hearing in the right ear to be abnormally acute.

General, laboratory and roentgen examinations gave negative results.

Examination of the eyes showed them to be normal externally. The pupil of the right eye did not react directly to light. The vision in the left eye was 6/200. There was bilateral secondary atrophy of the optic nerve.

My interpretation of the sequence of events in this case is otitis media followed by thrombosis of the lateral sinus; extension of the thrombotic process, accounting for the conjugate deviation of the eyes; extension to the cavernous sinus and surrounding structures, resulting in increased intracranial pressure and the symptoms of diabetes insipidus; and, finally, through necrosis of the hypophysis, the development of Fröhlich's syndrome.

THROMBOSIS OF THE LONGITUDINAL SINUS

The primary form usually commences in the middle fifth of the superior longitudinal sinus (Byers and Hass¹⁰). The high position of the sinus, low pressure, slow current and the presence of pacchionian bodies predispose to it (Wohllwill¹¹). It occurs usually in debilitated infants and as a result of actual changes in the blood itself, notably in chlorosis. If the obstruction of the sinus is considerable, it leads to hemorrhagic extravasations into the cerebral cortex, with subsequent softening, increased intracranial pressure and blood in the spinal fluid.

Some of these patients have symptoms indicative of cortical irritation. Jacksonian convulsions occur frequently. As the superior external surface of the cortex in the motor region for the lower limbs is involved, symptoms of involvement of the pyramidal tract confined to

10. Byers, R. K., and Hass, G. M.: Thrombosis of Dural Venous Sinuses in Infancy and in Childhood, *Am. J. Dis. Child.* 45:1161 (June) 1933.

11. Wohllwill, Friedrich, in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 6, p. 17.

the lower limbs may be present (Holmes and Sargent¹²). Papilledema and engorgement of the vessels of the scalp, retina and conjunctiva occur, but, on the other hand, there may be a complete absence of signs when subsequent autopsy observations make it difficult to understand their absence. Conjugate deviations of the eyes occur often. Exophthalmos and optic neuritis have been reported rarely. Uthoff referred to Good's case of cortical blindness.

When the thrombotic process is septic and has extended to the longitudinal sinus from the lateral sinus, the prognosis for life is poor. Doyle¹³ in describing this type concluded: (1) In the absence of meningitis, early apathy or stupor in a patient with evidence of thrombosis of the transverse sinus secondary to otitis media indicates infectious thrombosis of the superior longitudinal sinus by retrograde extension, especially if associated with choked disks or convulsions; (2) when tumor or inflammatory disease can be excluded, jacksonian seizures showing progression from one foot to the other or beginning in the foot and gradually involving the homolateral upper extremity suggest impairment of the circulation of the cerebral veins and probably thrombosis of the superior longitudinal sinus, and (3) abrupt onset of symptoms of increased intracranial pressure with fluctuations suggests thrombosis of the superior longitudinal sinus as well as ventricular tumor. The absence of the element of progression after a fair length of time or actual regression of symptoms is suggestive of thrombosis of the superior longitudinal sinus. If the syndrome is associated with edema of the lids and dilatation of the veins of the eyelids and forehead, and if fracture of the skull, orbital and periosteal infections and thrombosis of the cavernous sinus can be excluded, the diagnosis is established.

PAPILLEDEMA IN THROMBOSIS OF THE LATERAL SINUS

Bilateral papilledema occurs frequently in thrombosis of the lateral sinuses and does not necessarily indicate a poor prognosis. Dill and Crowe¹⁴ observed it in twelve of their thirty cases of thrombosis of the lateral sinuses. In ten patients the papilledema was bilateral, and in two it was unilateral. They attributed its bilateral occurrence to increased intracranial pressure and its unilateral occurrence to involvement of the cavernous sinus.

Benedict has stated that bilateral papilledema in these cases signifies increased intracranial pressure but does not indicate its cause. Abscess

12. Holmes, C., and Sargent, P.: *Brit. M. J.* 2:493, 1915.

13. Doyle, John B.: *Obstruction of the Longitudinal Sinus*, *Arch. Neurol. & Psychiat.* 18:374 (Sept.) 1927.

14. Dill, J. Lewis, and Crowe, S. J.: *Thrombosis of Sigmoid or Lateral Sinus: Report of Thirty Cases*, *Arch. Surg.* 29:705 (Nov.) 1934.

of the brain and meningitis are frequent causes. In other cases it has been assumed that the papilledema is due to "serous" or "protective" meningitis or to "otitic hydrocephalus." These descriptive titles have not had a satisfactory anatomic and pathologic basis. It is in this connection that Woodhall's¹⁵ recent work is of interest.

RECENT INVESTIGATIONS

The investigations carried out by Dr. Barnes Woodhall had to do with the relationship between increased intracranial pressure and thrombosis of the intracranial venous sinuses. The work is valuable in that it appears to establish a reasonable explanation for the occurrence of bilateral papilledema in cases of lateral sinus thrombosis and tends to indicate that in certain cases so-called serous meningitis may result from thrombosis of the dural sinuses.

Previous investigators have established wide variations in the pattern of the intracranial venous sinuses. These variations occur most frequently in the region of the torcular and have already been described. Woodhall, by examining two hundred autopsy specimens, has confirmed previous observations. Reports in the literature indicate that these variations are of importance. Among others Linser,¹⁶ Kummer¹⁷ and Eagleton¹⁸ have reported cases in which at autopsy thrombosis was found to be associated with abnormalities of the unaffected sinuses. Bilateral sinus thrombosis or unilateral sinus thrombosis, when a great disparity in the size of the lateral sinuses exists, has been observed to result in increased intracranial pressure. Recently, abnormalities of the sinuses have been reported as responsible for negative or false positive readings in the Tobey-Ayer modification of the Queckenstedt test as employed in the diagnosis of thrombosis of the lateral sinuses. These clinical observations, verified by the occurrence of similar cases during the period of these investigations, have led to the conclusion that inadequate venous drainage results in increased pressure. Ordinarily the lateral sinus of the opposite side can take care of the extra volume of blood when one lateral sinus is thrombosed, but if there is not an adequate cross-circulation or if the unobstructed lateral sinus is small there is resultant back pressure and consequently an increased intracranial pressure. It is suggested by Woodhall that this is the anatomic basis

15. Woodhall, Barnes: Variations of the Cranial Venous Sinuses in the Region of the Torcular Herophili, *Arch. Surg.* **33**:297 (Aug.) 1936. Woodhall, B., and Seeds, A. E.: Cranial Venous Sinuses, Correlation Between Skull Markings and Roentgenograms of the Occipital Bone, *ibid.* **33**:867 (Nov.) 1936.

16. Linser, P.: *Beitr. z. klin. Chir.* **28**:642, 1900.

17. Kummer, E.: *Rev. de chir.* **19**:531, 1899.

18. Eagleton, Wells P.: *Arch. Otol.* **25**:91, 1906.

for bilateral papilledema as observed in acute thrombosis of the lateral sinus and also in certain cases described as instances of serous meningitis when there is a history of an otitic infection and a sinus block can be demonstrated.

In addition to the verification of previous observations and correlation of them clinically, a technic for obtaining roentgenograms of the occipital bone enabling estimation of the size of the lateral sinuses has been worked out.

CONCLUSIONS

The ocular signs of thrombosis of the cavernous, lateral and superior longitudinal sinuses are briefly reviewed.

Necrosis of the hypophysis was noted in five of the six cases reported. An additional report illustrated the possibility of hypophysial involvement through thrombosis of the lateral sinus.

Recent work is briefly summarized. It appears to supply an anatomicopathologic basis for the occurrence of bilateral papilledema in thrombosis of the lateral sinus and in certain cases of so-called serous meningitis. The work requires confirmation.

DISCUSSION

DR. THOMAS H. JOHNSON: The subject of thrombosis of the intracranial venous sinuses is an important one to the ophthalmologist. Early in the onset his opinion may be the deciding factor in the diagnosis. The society is fortunate in having the subject so ably presented by Dr. Walsh.

I have not observed any case of aseptic sinus thrombosis in which a diagnosis was made ante mortem. I shall limit my remarks to a discussion of septic thrombosis. In retrograde thrombosis the ocular symptoms are less severe and come on later than when the infection has been anterior. The explanation for this lies in the anatomy. The course of the infection in anterior thrombosis is along the ophthalmic veins, which traverse the orbit on their way to the cavernous sinus. The orbital tissues and cavernous sinus therefore become involved earlier in the anterior infections than in the retrograde infections.

In my experience swelling of the upper eyelid in orbital and intraocular infections has been more common than swelling of the lower lid and is very prevalent in such infections.

Exophthalmos is an important early sign, whether pus is present in the orbit or not. It is due to an orbital cellulitis; probably venous stasis is also a factor. In three of Dr. Walsh's cases there was an abscess in each orbit, and in one an abscess in the orbit on the side of the mastoid infection. There may be marked exophthalmos without the presence of pus. I have in mind two cases that I observed in consultation. One patient was a young woman who had squeezed a small pimple on the right upper eyelid. The next day she had exophthalmos on the right side, edema of the conjunctiva and a slight blurring of the margin of the disk. The exophthalmos rapidly increased, and an incision was made into the orbit through the upper and lower nasal fornices and drains inserted. I saw her on the follow-

ing day; the papilledema had increased, she had a high temperature and there was no drainage from the orbit. She died the next day. The other patient was a young medical student who, after having a tooth extracted, acquired unilateral exophthalmos with orbital cellulitis. There was slight papilledema on the involved side. Deep incisions in the orbit revealed no pus. The termination was fatal. There is slight doubt that the orbital cellulitis is a result of an infection and that if the patient should survive long enough pus would appear in the orbit.

The nerves that lie in the cavernous sinuses are exposed to toxins and pressure from the thrombus which lead to paralysis, but the edema in the orbital tissues is also a factor in producing complete ophthalmoplegia externa. When the lateral rectus muscle of the opposite eye is involved, I think that it is an indication that the process has extended to the other cavernous sinus or that intracranial pressure has developed. It is common in instances of tumor of the brain to see paresis of the lateral rectus muscle from intracranial pressure. Anesthesia of the cornea and the skin of the forehead and cheek may be produced by pressure in the cavernous sinuses on branches of the fifth nerve.

I do not agree with Dr. Walsh that ophthalmoscopic signs are of slight importance in making a diagnosis in thrombosis of the cavernous sinus. When present, edema of the head of the nerve is of great diagnostic importance. Manifest edema of the head of the nerve has been present in the early stages in most of the cases I have observed. When bilateral papilledema is present, I think that it indicates that the infectious process has extended to the lateral sinuses or has set up a serous meningitis or abscess of the brain with a concomitant intracranial pressure. The intracranial pressure may be due to inadequate venous drainage, complicating serous meningitis or abscess formation. Therefore, bilateral papilledema has important prognostic significance, as it indicates an extension of the process and intracranial complications, as does also paralysis of the contralateral sixth nerve. The edema of the intracranial tissues is probably analogous to that of the orbital cellulitis.

An early appearance and severity of the ocular symptoms probably indicate that the infection is anterior rather than retrograde.

DR. R. TOWNLEY PATON: It is indeed a pleasure to participate in the discussion of so interesting a subject as the ocular signs of thrombosis of the intracranial venous sinuses.

After reviewing the literature carefully, I find that there is little for me to add in criticism or as an explanatory note to the first portion of this paper. No mention is made of the pitfalls to be avoided in making a differential diagnosis and etiologic diagnosis of thrombosis of the intracranial venous sinuses. Most authors agree that malaria, typhoid, bronchopneumonia and erysipelas are often overlooked until late in the disease. Also in passing, I should like to mention the Crowe-Beck sign, which is little known to ophthalmologists but which has a definite clinical value. Crowe stated that "if in a normal person one internal jugular vein is compressed with the finger, no appreciable evidence of stasis is to be seen in the supra-orbital or retinal veins. If, however, both internal jugulars are compressed at the same time, a marked dilatation ensues. If the pressure is now released on one side, while it is maintained on the other, the engorged veins empty immediately. If, however, the jugular bulb is thrombosed on one side—say

the right—the veins will still remain engorged if the pressure is maintained on the left internal jugular, even though it is released on the right side.” I should like to ask Dr. Walsh the value of this sign.

The second part of the paper, dealing with the autopsies and complete records of six cases, shows how carefully this subject has been studied. Dr. Walsh points out that the hypophysis is frequently involved—a point not emphasized by previous investigators.

Dr. Walsh made a statement that inadequate venous drainage results in increased pressure. I suppose that he is referring to increased intracranial pressure. No mention was made of the effect of intra-ocular pressure or whether the intra-ocular pressure was different in the cases of unilateral involvement. Lauber states: “All observations and experimental facts support the idea that increase of diastolic venous pressure in the retina and its relation to the diastolic arterial pressure in the retina are decisive for the formation of papilledema. If the arterial tension in the retina is low, even a slight increase of venous pressure caused by high intracranial pressure produces papilledema.” It would seem, then, that so long as one does not know the intra-ocular pressure, as measured with a tonometer, one’s interpretation of the underlying etiologic conditions may be wrong. This point then remains to be proved, but the inference of Dr. Walsh that in cases in which there is bilateral papilledema the condition is the result of a compensatory mechanism due to a congenital malformation of the lateral sinus is suggestive, and it is the first time that the clinical and pathologic investigations have been so thoroughly discussed.

DR. FRANK B. WALSH: Increased corneal sensitivity and corneal anesthesia in thrombosis of the cavernous sinus are due to involvement of the ophthalmic division of the fifth nerve. It has been suggested that pain in the face and behind the eye in petrositis may be due to involvement of the vidian rather than the fifth nerve. This seems unlikely, owing to the fact that the sphenopalatine ganglion supplies branches to the palate and to the nose, to which pain is not referred.

I do not wish to minimize the importance of ophthalmoscopic signs in thrombosis of the cavernous sinus, but I believe that considered singly they are of slight value in arriving at a diagnosis, as they usually occur late. Furthermore, bilateral or unilateral papilledema occurs frequently in thrombosis of the lateral sinus, when there are no other signs that would indicate involvement of the cavernous sinus.

The possibility of changes in the intra-ocular pressure has not been considered in this paper.

Woodhall’s work seems to establish a definite basis for the occurrence of papilledema in thrombosis of the lateral sinus as well as for so-called serous meningitis when there is a history of otitis media and a sinus block can be demonstrated.

My attempts to demonstrate the Crowe-Beck sign have been unsuccessful.

I wish to thank Dr. Johnson and Dr. Paton for their discussion.

A SECOND GROUP OF CASES OF ARACHNODACTYLY

RALPH I. LLOYD, M.D.

BROOKLYN

Two years ago, at a meeting of this society, I reported six cases of arachnodactyly in two families. Through the courtesy of the surgeons of the Brooklyn Eye and Ear Hospital and their assistants, nine more cases will be added. The new cases have not explained the origin of the condition, but hereditary influences are more certain. Perhaps the best way to present this matter will be to describe the new cases and summarize briefly the points that they emphasize.

REPORT OF CASES

CASE 1.—David B., aged 15, from Dr. Place's clinic, had vision of counting fingers at 6 feet (1.8 meters), but by holding print close to an eye with a little study he could read ordinary print in a surprising manner. Both corneas were smaller than normal, but I did not measure them. The tension (according to the new Schiötz scale) was 18 mm. in one eye and 13 mm. in the other. The left lens was dislocated. It was hard to see the other lens, but it was probably dislocated also. This element of uncertainty was due to the fact that the pupils did not dilate, even after three days of treatment with a 1 per cent solution of atropine instilled twice daily. There was an opacity in each lens like a rider, from below, and one adhesion in the right eye at 7 o'clock, which was not explained by anything in the patient's history. In the pupillary area of each eye were remnants of pupillary membranes, and in the lenses beneath were numerous opacities. The adhesion was probably one of these remnants. The patient had curved little fingers, a depression in the midline of the lower part of the sternum and pulmonary stenosis (G. H. Roberts), which was evidently not serious. The height was 59 inches (1.5 meters), and the span, 68 inches (1.7 meters). He had three normal sisters, and the parents' eyes were normal (so the sister said), although the father was tall and thin. The parents and two sisters could not be induced to come to the clinic, but the sister who brought the patient was normal in every respect. Of course, the fundus could not be inspected, because of the small pupils; the miosis was the most stubborn of any I have seen. The transmitter of the condition was evidently the father, but as my associates and I have never seen him, the question remains open. The patient's skeletal changes were not extreme but were typical of arachnodactyly, and there was also pronounced lordosis. This patient showed some of the characteristics of status dysraphicus, such as curved little fingers, a depression in the sternum and lordosis, but the exaggerated span, the heterochromia and the syndrome of Horner were lacking. The patient was of German ancestry. The father was an uncle of the mother. The patient gave a negative

Read at the meeting of the American Ophthalmological Society, Hot Springs, Va., June 1, 1936.

Wassermann reaction. This is the only consanguineous marriage in the history of any of the patients I have seen; also, this patient is the only one of either group that I have studied with small corneas. Megalocorneas have been reported, but I have not seen any.

CASE 2.—Dr. Irving Jacobs had among his private patients a boy of 7 of Italian parentage. This patient was of the same general build as the patient in case 1 but would escape inclusion in this group but for his eyes. The father had congenital ptosis of the left upper lid, but the mother and another boy and one

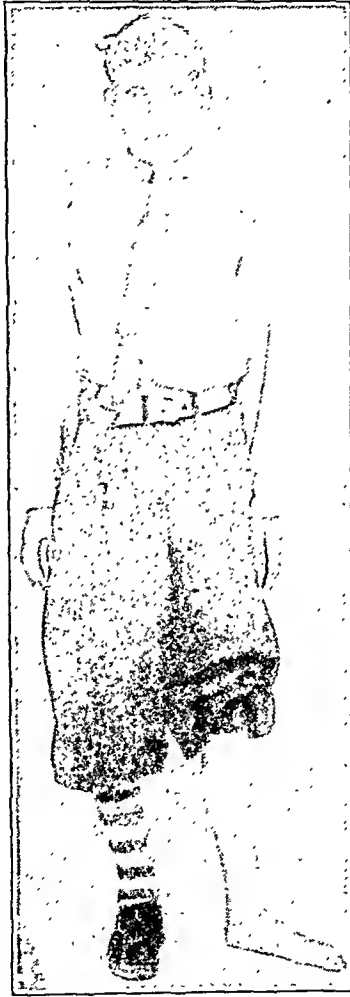


Fig. 1 (case 1).—Photograph of the patient, showing typical arachnodactyly associated with congenital heart disease.

girl are normal. Vision was counting fingers at 5 feet (1.5 meters) in both eyes, and skiascopy showed 14 diopters of myopia. The pupils dilated well under the influence of atropine, and both lenses then appeared much smaller than normal, with a clear margin all around except above inward (fig. 2). Slit lamp examination made it plain that the anterior surfaces of the lenses were almost round. The lenses were not entirely clear. Owing to the reduced diameters, the impression that one received first was that the lenses were pushed back rather than displaced laterally. Visual acuity could be improved to 10/200. The fibers of the zonules were long and unbroken,

but the lenses were tremulous. Some months later the right lens was dislocated into the anterior chamber, with the characteristic symptoms that go with that condition: The upper pole of the lens was a little behind, and the lower pole somewhat in front of, the iris. The spherical outline of the lens was evident now more than ever. Vision was limited to perception of light, but projection was good. A strong beam of light was thrown on and into the eye by Dr. Jacobs, and the iris suddenly contracted and forced the lens back into the vitreous, but it came back into the anterior chamber when the patient sat up. Pilocarpine was used for the purpose of contracting the pupil behind the lens and holding it there to make extraction easier. But, as in the history of patients whose cases have been reported elsewhere, the tension rose to 45 (Schiötz), and pain and congestion were immediately aggravated. All the symptoms were relieved when the miotic was discontinued. The next day Dr. Jacobs removed the lens, which had the classic globular form to be expected when the fibers of the zonule are relaxed or broken, but it seemed entirely clear to the naked eye, as a normal lens should, which was a different finding from that indicated by the slit lamp examination. Prolapse of iris tissue into the wound required making a conjunctival

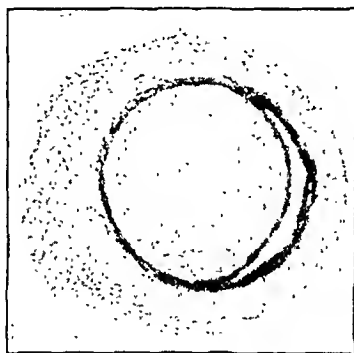


Fig. 2 (case 2).—Drawing of lens, showing the spherophakia of 14 diopters. The parents and two other children were normal.

flap a few days later, but recovery was complete, without any other complication. The lens was removed from the eye without difficulty, and there was no tendency for the lens to fall back into the vitreous. On recovery the patient accepted a +10.50 diopter sphere and a +2.25 diopter cylinder, axis 170, with resulting visual acuity of 20/50. The usual refractive value of the lens amounts to about 10 or 12 diopters, but in this case there was a difference of 25 diopters, indicating that the globular form of the lens, and not the lengthening of the eyeball, accounted for the myopia. My associates and I suspected that this patient was mentally defective. It might be mentioned in this connection that congenital dislocation of the lenses among patients with mental deficiency has been observed more frequently in Switzerland than elsewhere. The second lens was now more freely movable than it had been, and its complete dislocation was expected. (This paper was read early in June. Late in the same month the expected dislocation of the left lens into the anterior chamber occurred. No miotic was used, but the symptoms were easily controlled by atropine. The lens was easily removed and weighed 0.157 mg. It measured 7 mm. in both the vertical and the horizontal diameter, and from the anterior pole

to the posterior pole it measured 6 mm. At the anterior pole was a trace of the flattening found in the normal lens, and the lens seemed as clear to the naked eye as the first one removed.)

CASES 3 and 4.—A girl 9½ years old came from Dr. Ohly's clinic complaining of poor vision (fig. 3). Vision was improved to 20/70 with a +10 diopter sphere +2 diopter cylinder, axis 105 for one eye and a +8 diopter sphere +2 diopter cylinder, axis 75 for the other. Both lenses were dislocated to such an extent that the pupillary areas were almost entirely freed. The right lens was displaced

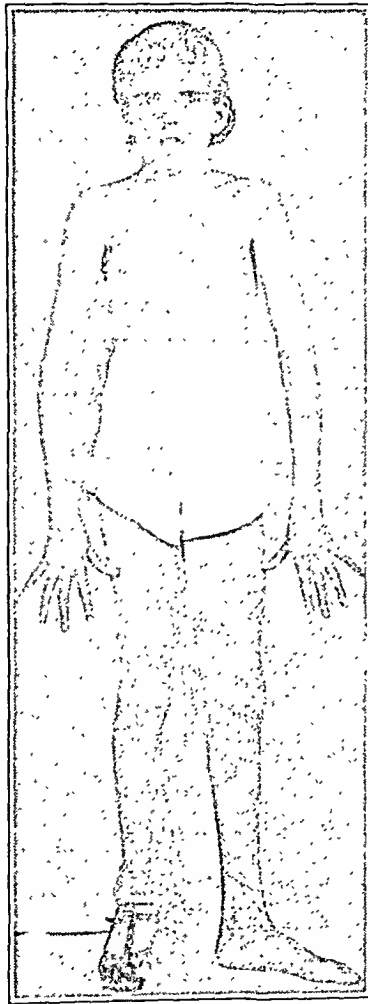


Fig. 3 (case 3).—Photograph of the patient, showing typical arachnodactyly. The father was normal; the mother had mild ectopia lentis but none of the skeletal changes of arachnodactyly.

upward and outward, while the left was displaced almost directly upward. From the lower border of each lens broken strands of fibers of the suspensory ligaments waved freely. The patient showed typical arachnodactyly. The father and two children (aged 8 years and 2½ years, respectively) were normal. A 9 month old child required a very large shoe, but no other signs of the ailment could be found. Under the influence of atropine the patient's pupils opened about half the normal extent. The mother was plump and short and was pregnant at this time. The mother's eyes were of much interest, since the occurrence of incomplete syndromes

has been recognized. She wore a +5.50 diopter sphere +2.50 diopter cylinder, axis 90 for one eye and a +7 diopter sphere +0.50 diopter cylinder, axis 30 for the other, with visual acuity of 20/50 for three letters and 20/20 for three letters, respectively. Her pupils did not dilate well after the instillation of homatropine, but the right lens was dislocated upward and inward and the left lens was somewhat out of position. The right eye turned in when she was small and was operated on at the age of 19, with a good, but not a perfect, cosmetic result. In neither eye could margins of the lens be seen, but the dislocation was evident from the shadow seen at the bottom of the dilated pupillary area.

In the various families under observation a parent with dislocation of the lenses but a great deal of subcutaneous fat and no bony changes has had children with the complete syndrome, and a parent with normal eyes but pronounced bony changes and no subcutaneous fat has also produced children just as typically affected. From descriptions in the literature and from findings in the groups of cases reported my associates and I have come to regard congenital bilateral dislocation of the lens as a part of a syndrome which, when complete, is one of typical arachnodactyly, but either dislocation of the lens or the skeletal changes may be missing. There is no exact standard to enable one to distinguish the slight departures from the normal skeleton and musculature; there is no doubt that slight dislocations do escape detection, but any great departure in this respect would eventually be found, especially if there are other cases of ectopia lentis or suspicious skeletal changes in the preceding or following generations. The mother was considered to be the transmitter in this family, but it was impossible to obtain reliable information about the preceding generation.

CASES 5, 6, 7 and 8.—A girl of 7½ years from Dr. Ohly's clinic was brought to the hospital by her mother because of poor vision. This child had been very deaf from birth, and both lenses were displaced upward and inward. The lenses were clear on ophthalmoscopic examination, but slit lamp examination disclosed many fine dots and some haze. Vision with a -6.00 diopter sphere for one eye and a -5.00 diopter sphere for the other was 10/40—3 letters and 10/70, respectively. The department for patients with diseases of the ear found nothing to explain the deafness, and there was no history of illness in infancy to account for it. There was a small crescent of clear pupil in each eye, but the vision obtained was through the lens. The pupils dilated well; the long fibers of the suspensory ligaments were clean and free from deposits of pigment, and no ruptured strands were found. The irides trembled, but the lenses did not. The body showed the changes typical of arachnodactyly, and there was roughness of the pulmonary first sound. Divergent strabismus was present in the eye with the poorer vision. The older sister was reported as having had similar signs, including deafness, poor vision, delayed dentition (from ten to twelve months) and lateness in walking. She was also tall and thin, and a lantern slide shows the strong resemblance to the affected parent. This child was killed in an automobile accident. She wore a +7.75 diopter sphere for both eyes, but the clinical record gave no other details, as she was very young at the time of examination. The mother

was not affected and had normal vision with a -2.00 diopter sphere for one eye and a -3.00 diopter sphere for the other. The father was the transmitter, and he evidently derived the influence from his father, as figure 4 C shows. The father of the child wore a -4.00 diopter sphere for the right eye and a -2.00 diopter sphere -6.50 diopter cylinder, axis 120 for the left, with visual acuity of 5/200 and 20/33 (dif.) with this correction. The correction for the right eye was merely a "gesture," as it did not influence the vision at all, and there was so much astigmatism that it could not be measured. The right lens was displaced outward, and about half of the pupil was free from the lens. The left lens was

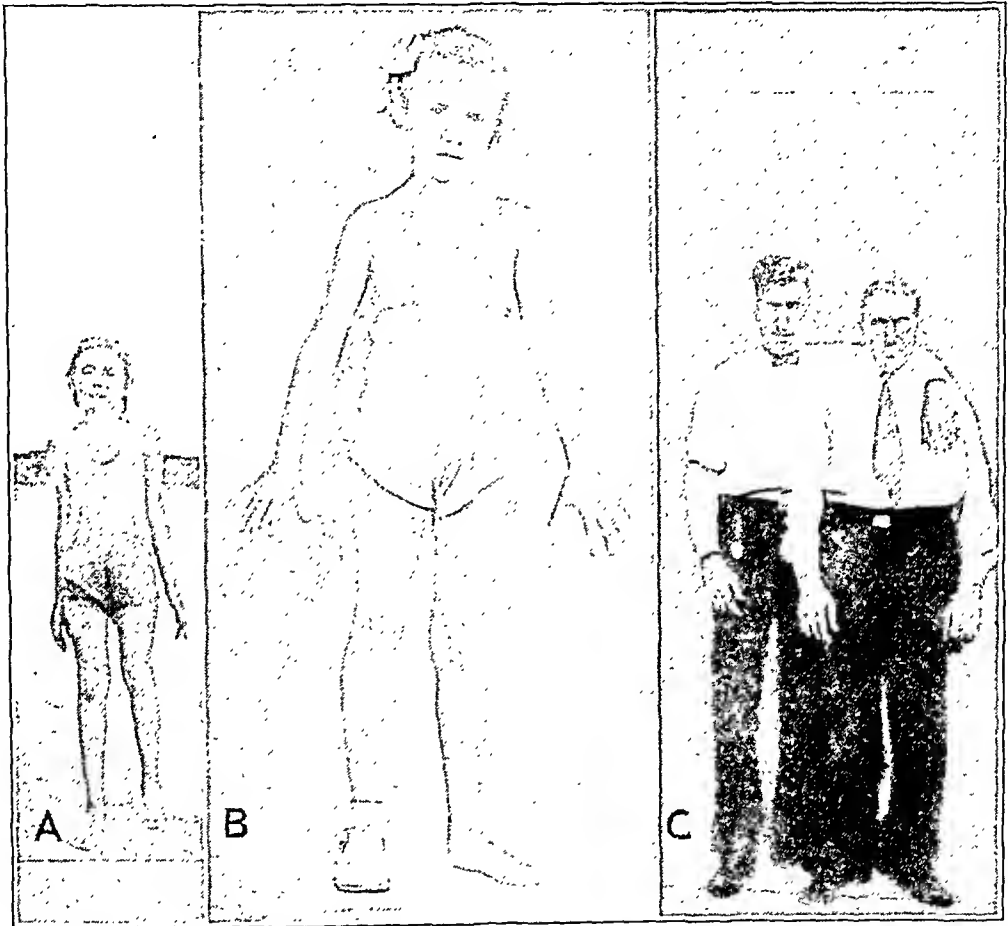


Fig. 4 (cases 5, 6, 7 and 8).—A, photograph of the older sister of J. K., deceased. B, photograph of J. K., showing typical arachnodactyly; vision with correction was 10/40 in one eye and 10/70 in the other. C, photograph of the father, also showing typical arachnodactyly, with his father beside him. The latter had not been examined.

displaced but little; it was rounder than the usual lens, and there were two notches in its border. The suspensory fibers were in good condition, very long and clearly seen, with dots of pigment adhering to them here and there, and there were numerous minute opacities scattered throughout the lenses. The patient showed typical arachnodactyly, and the photograph of his father, who also had a history of poor vision, indicates clearly the line along which the inherited disorder was transmitted.

In this connection it is interesting to note that none of the patients of the two groups have been blonds. Some were very dark skinned and the hair was very black, despite the light hair of the unaffected parent. None of the patients seen has been of English ancestry. One was of German origin; two families were Jewish, and the rest have been Italian.

The last case is the most remarkable.



Fig. 5 (case 9).—Photograph of the patient. The age was 14 years; the weight, 165 pounds; the height, 6 feet, 8½ inches; the span, 6 feet, 11½ inches, and the weight at birth, 10½ pounds.

CASE 9.—The patient was sent to me by Dr. Ohly from his private practice. The mother was not affected, but no satisfactory information about the father could be obtained. Inquiry into his physical make-up and character was skilfully deflected. The patient was 6 feet, 8½ inches (2.04 meters) tall at the age of 14 years, and he had a span of 6 feet, 11½ inches (2.1 meters). His body showed the changes typical of arachnodactyly, but physical examination and an electrocardiogram (G. H. Roberts) showed no abnormality. The Wassermann

reaction was $+-$, and there were bilateral reducible hernias of no importance. Because of his height, a roentgenogram of the skull was taken; the pituitary gland and the clinoid processes were normal. Dr. Rendich reported some indications of increased cerebral pressure. The weight was 165 pounds (74.6 Kg.). The weight at birth was $10\frac{1}{2}$ pounds (4,762.7 Gm.). The pupils were very small and dilated little; the irides shook. The right eye showed much vitreous opacity, with many soft light-colored spots in the choroid and thinning between the choroidal vessels. The outlines of the disk were not clear; the disk seemed somewhat swollen, but the view was not good enough to enable one to be positive about these finer details, because of the small pupils and the hazy vitreous. The right lens could not be seen. The left lens was not clear, and its entire anterior surface was covered with a fine network of remnants of persistent pupillary membrane



Fig. 6 (case 9).—Photograph of the patient as an infant. Note the right thumb.

composed of triangular spots with fine tails running from the angles. The left lens was tipped far back, retaining an attachment below. No comment about the patient's body formation or lack of subcutaneous fat is necessary, as figure 5 shows the details. The slit lamp showed long strands of shreds of persistent pupillary membrane that floated backward and forward through the pupils (when these were dilated), especially in the left eye. They reminded one of a wisp of cotton moving in a draft that is too weak to affect any other thing. The movement of the shreds backward through the pupil into the vitreous and then forward through the pupil into the aqueous was the result of the pressure of the upper lid as it descended in winking and the release of this as the lid rose. The patient wore a size $11\frac{1}{2}$ shoe, and his fingers, elbows and wrists could be bent back so far that the appearance was uncanny. The photograph of the patient as an infant (fig. 6) shows this feature in the thumb of the right hand. This abnormal flexi-

bility of the fingers may turn out to be a reliable early sign of the skeletal changes which can vary within such wide limits without attracting attention unless the vision is poor. The scapular angles were not everted, but the right shoulder drooped, and the right scapula was pushed backward. The patient's father and grandfather were said to have had poor eyes, but no other details of the histories of these ancestors could be learned. The abnormal height of this patient suggests pituitary hyperactivity, and other observers have reported their suspicions of a disorder of the pituitary gland, but no one seems to have found pituitary involvement.

Dr. L. G. Rowntree, of Philadelphia, has made experiments on animals with extracts of pineal and thymus gland and has speeded up the development of white rats in an amazing manner with injections of thymus extract, while pineal extract retarded development of the skeleton. This is a line of thought worth following, but it hardly seems possible to expect anything of any remedy in this patient at this age. Perhaps in a family in which there was a typical case of this condition in a parent, treatment along the line mentioned might aid normal body development of a young child or prevent or mitigate extreme clouding and displacement of the lens. In some cases the dislocation of the lenses seems to become steadily worse, and most of the dislocations into the anterior chamber have occurred in rather young patients.

COMMENT

Spherophakia and microphakia appear to be advanced phases of congenital dislocation of the lens, due to unusual and progressive laxity of the suspensory ligament. This permits the natural elasticity of the lens to produce an apparently small but almost globular lens. The vertical and horizontal diameters are necessarily reduced, and eventually dislocation of the lens into the anterior chamber follows. Assuming that congenital dislocation of the lenses is but a part of a syndrome and not an entity by itself, I have looked over the literature on cases of this condition reported during the past twenty years and have found a number of instances in which congenital heart disease was reported. Also, the condition has been found to be more frequent among young inmates of institutions for the feeble-minded. None of the reports of cases of bilateral congenital dislocation of the lens have mentioned skeletal defects to justify the idea that the signs of arachnodactyly have been observed in the past but only the changes in the lens reported. Nevertheless, I am sure that the syndrome has escaped observation.

Some of the patients with the complete syndrome observed have shown numerous pigmented clumps adherent to the fibers of the suspensory ligaments, and marked choroidal changes have also been found, even in children. Can one not assume, then, that high myopia

which is strongly hereditary, with its attendant changes in the fundus and the elongation of the eyeball behind the equator, may be due to abnormal weakness of the fibers of the suspensory ligament, allowing the lens to assume a more spherical form accounting for the early myopia, while the changes in the fundus of myopia could be explained as effects of the choroidal disease indicated by the deposits of pigment on the suspensory fibers and the changes in the tapetum?

No satisfactory explanation for arachnodactyly has been given. Weve's theory that the disease is a mesodermal dystrophy satisfies most objections, but not all. The dilator fibers of the iris arise from another layer of the embryo, but they may be ineffective through no error of their own structure but only because the framework of the iris is unyielding. But how can one explain the abnormal weakness of the suspensory fibers that plays such an important rôle in producing the dislocation of the lens? They, too, arise from another layer of the embryo. Vogt suggested that the disease transmitted from one generation to another operates on the chromosomes in the ovum, and the syndrome is complete or partial as more or fewer of them are affected. This is a rather satisfying explanation, but Passow has reported a group of cases in which he pointed out certain features typical of syringomyelia but of a milder degree than is usual in that serious ailment. He argued that many of the symptoms common to arachnodactyly and syringomyelia are the result of improper formation of the neural tube at the stage when the ridge of tissue which connects the submerged neural tube with the surface splits up into two groups of cells on each side of the primitive spinal cord. From these groups of cells, on each side of the median line, one row of cells migrates forward and laterally to form the chain of sympathetic ganglions on either side of the midline, anterior to the spinal column, and the other series forms the chain of ganglions on the posterior sensory nerve roots. In extreme cases the cells surrounding the neural canal are assumed to be affected also, and as their functions are of a trophic nature many of the symptoms characteristic of syringomyelia are explained. In patients with milder syringomyelic symptoms, one sees the changes characteristic of arachnodactyly and also such signs of trophic disturbance as heterochromia and Horner's syndrome, which are disturbances of the sympathetic system. Changes such as abnormal length of the arms (so that the span is greater than the height) and long, thin bones and weak muscles are considered to be trophic. The hands may be very large and their integument moist and soggy. In females, the breast on the side affected by the heterochromia or the Horner syndrome may be much smaller than the opposite breast. Spinal curvatures are common in any type of arachnodactyly. The fingers,

instead of being straight, may be curved somewhat, though the curvature never reaches the degree of the clawhand of patients with syringomyelia. In the minor degree of curvature of this type found in arachnodactyly, only the little finger may retain the aforementioned curvature. Another defect found in the patients in whom the condition is of the syringomyelia type is a sternal groove so deep in some cases that the heart is displaced to the left. Some of these features have been found in the cases reported in this paper, except the syndrome of Horner and the heterochromia. Most of the patients with acquired heterochromia whom I have seen showed evidences of tuberculous uveitis, but now and then a notable exception appeared. Unfortunately, my acquaintance with this aspect of arachnodactyly is so recent that heterochromia has not been studied from this angle.

To some a study of a rare condition of this kind seems profitless. With this view I do not agree. One does not know where study of this condition will lead or what other conditions may be involved in the process producing this unique disorder. Some do not care to study unusual conditions holding no hope of cure. If either of these attitudes is accepted one must not expect another discovery like that of insulin in the field of medicine. At present there is no satisfactory method of finding cases of atypical arachnodactyly unless the patient has the characteristic dislocation of the lenses. That patients with atypical arachnodactyly without ocular symptoms transmit the condition is undeniable. Also, patients with bilateral dislocation of the lenses without body changes become transmitters of the complete syndrome. The practical technic of finding cases of the complete syndrome at present is to work backward or forward in the family of any patient with congenital bilateral dislocation of the lenses. Pedologists do find patients with this condition without the ocular involvement, but it is not known how many patients with mild arachnodactyly without lenticular changes escape detection under the present procedure.

If one fixes firmly in the minds of chiefs of clinics and their associates the importance of the ocular symptoms as an index of this condition, more cases will be found; families with small children can then be studied, and perhaps the condition in the younger patients can be controlled. Whatever the outcome and however rare the condition, this problem is a challenge, and although the more common and serious ocular ailments properly preempt a larger part of the clinician's time and attention, nevertheless there should still be time enough for each to contribute something toward the solution of the problem from the clinical side. As in other questions involving heredity, the laboratory should give important if not decisive information.

A bibliography will be found with my first article, which was presented before the American Ophthalmological Society.¹

Another bibliography will be found with the very excellent article on this subject by Dr. Frank Burch² which was read before the Section on Ophthalmology at the Eighty-Seventh Annual Session of the American Medical Association in June 1935. The only other article published recently is that of Bakker.³

1. Lloyd, Ralph I.: Arachnodactyly (Dystrophia Mesodermalis Congenita; Typus Marfanis; Marfan's Syndrome; Dolichostenomelia), Arch. Ophth. **13**:744 (May) 1935.

2. Burch, Frank: Association of Ectopia Lentis with Arachnodactyly, Arch. Ophth. **15**:645 (April) 1936.

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EXAMINATION AND CARE OF THE EYE IN RELATION TO LIGHTING

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A strong incentive for our work in physiologic optics has been to help prepare the way for a more extensive use of hygienic measures in the care and treatment of the eye. The subject of hygiene has been built up in the attempt to preserve and restore normality of function through regulation of the conditions under which the organ functions. In relation to the care of the organs of the body the growth of hygiene has been rapid. In relation to the sense organs, however, its progress, it would seem, has suffered perhaps undue retardation. Whether this situation is intrinsic or merely marks a stage in the development of the subject time alone can tell. In the case of the eye, at least, it would seem that hygiene can be made to play a much more important part than it now does, that is, that the care and treatment of the eye shall not be so narrowly dependent on the surgical measures of operation and on the use of lenses and prisms.

IMPORTANT STRUCTURAL AND FUNCTIONAL CONSIDERATIONS

In connection with the hygienic treatment of the eye one must consider the muscular equipment of the eye, the structure and parts of the eye itself and the sensorium.

A notable feature of the visual mechanism is the delicacy and intricacy of its muscular equipment, and an important factor in the correctness of the visual performance is the normality of structure and function of this equipment. There are two aspects of hygienic treatment relating to this muscular equipment: (*a*) the correction of the gross and minor imbalances through either suitable exercises or other means as yet poorly defined and (*b*) the prevention of harm to the eye caused by malfunctioning of the muscles due to improper conditions of work. The influence of the grosser abnormalities on the accuracy of the combination of the images of the two eyes in binocular vision has received due recognition and study, but the influence of the lesser abnormalities on the clearness and correctness of the formation of these images has not perhaps been fully realized. One has only to remember how pliable

From the Research Laboratory of Physiological Optics.

the eye is to understand how readily it may be deformed by slight imbalances in the pressure exerted by the ocular muscles in holding it in proper balance in any given position or in executing accurately the many movements it is required to perform. When these pressures are excessive in amount or when there are special conditions of weakness of the coats of the eye, permanent changes in structure may be produced, or the deformities may be present only *in situ*, that is, only when the eye is subjected to the pressure and pull of the muscles.

In respect to these deformities it will be remembered that deviation toward a prolate spheroid will cause myopia; deviation toward an oblate spheroid, hyperopia, and irregularity in the deviation, astigmatism; that only a slight deviation is required to produce a diopter of defect, and that deformities may occur either in the ball of the eye or in the cornea. In relation to the influence of muscular pressure on the shape of the eyeball, the following points should be considered: the attachment of the straight muscles by a broad insertion well in front of the center of rotation of the eye and their passage around its curved surface to a point at the back and upper part of the socket or orbit, the passage of the two oblique muscles around the eye from their insertion at a point in the back toward the front of the orbit in such a way as to give to some extent the effect of suspending the posterior half of the eye in a noose, and the possibilities presented for excessive pressure or unevenness of pressure in the action of these muscles.

In this connection it should be borne in mind that the muscles have the twofold function of supporting the eye and of moving it and that it is in the relation or proportion of the forces which serve to support the eye that slight imbalances may be expected to occur. Slight disturbances in the relationship between the backward pull or tension of the rectus muscles and the opposing action of the oblique muscles, for example, may serve to change the length of the polar axis and otherwise deform the eye. Further, the correction of the imbalances in case of too strong a pull on the part of the converging muscles would require the action of the antagonistic muscles to prevent confusion of vision due to defective combination of the images of the two eyes, and this in turn might lead to an inequality of pressure which might serve to deform the ball of the eye or the cornea and thus tend to produce astigmatism, perhaps the customary astigmatism with the rule. Or again, in case of asymmetry in the pull or tension of these converging muscles this tendency toward torsion would have to be corrected by an opposing asymmetry in the tension of the antagonistic muscles. This composition of forces would tend to shift the direction of maximum pressure and produce oblique astigmatism.

It should further be remembered that these deformities as produced by the pressure of the muscles of the eye may be either congenital or acquired; perhaps they are most often acquired. It is, of course, in the cases in which the defect is acquired that there is the greatest possibility of success in the use of hygienic measures.

In relation to the pressures produced by the more customary movements of the eye and their tendency to cause elongation of the eyeball, it may be noted that the convergent movement considered from its beginning in what is called the primary position to its end, when the eye has made its maximum excursion inward, is a complicated function of three straight muscles, the internal, superior and inferior rectus muscles. The superior and inferior oblique muscles steady the eye during the excursion and perhaps aid the adduction in the more extreme positions of convergence. The diverging movement is also a complicated function of more than one muscle, the complication varying with the degree of abduction. In contracting, the converging muscles turn the front of the eye inward and press the eye against the external rectus muscle over a considerable portion of its length. The effect of the action of both sets of muscles, converging and diverging, is to exert a backward pressure on the contents of the eye toward the posterior pole, which backward pressure is opposed by the contraction of the oblique muscles. The point of attachment and the course of the oblique muscles, however, are such as to leave a segment of the back wall of the eye without external reinforcement. The front of the eye is reinforced by the pressure of the four straight muscles which are attached, as has already been indicated, on a broad base well toward the edge or limbus of the cornea and by the lens with its suspensory ligament. It is perhaps not too much to expect, then, that under the pressure of sustained convergence rendered excessive by bringing the work closer to the eye under poor lighting conditions and other faulty conditions of seeing the coats of the eye, particularly if they are already weak from congenital or from degenerative causes, should yield in the region of the posterior pole.

In further relation to the harm done to the eye by the malfunctioning of its muscles as a result of poor conditions of work the situation with regard to the accommodation of the eye should not be left out of account.

In the attempt to understand the effects of poor lighting on the eye one of the most fundamental principles to keep in mind is that the eye is always under a reflex incentive to clear up vision. This incentive is so strong that it is extremely difficult to oppose it successfully by an act of the will or voluntarily to force the eye to make an adjustment detrimental to clear seeing. The eye has grown up under daylight. Under this condition only three adjustments have developed, and, indeed, only three are needed: the reaction of the pupil to regulate the amount

of light entering the eye and to aid the lens in focusing the light from objects at different distances and accommodation and convergence to bring the object on the principal axis of the lens and the image on the fovea—conditions necessary for the formation of the clearest images by the lens and for the best discrimination of these images by the retina. These adjustments take place under a sort of triple bond imposed by their common nerve supply, which is so strong that if one takes place the others also take place unless the power of separating them has been acquired, and even then their separation is accomplished only by great effort or strain. Artificial lighting, with its unnatural and unfavorable conditions for clear seeing, has come late in the history of the race, and the eye has not developed any reactions or adjustments to meet the conditions imposed. Yet the incentive to clear up seeing remains and leads to adjustments which, if allowed to take place, serve only further to blur rather than to clear up vision. For example, a change in accommodation, which is a change designed to clear up an image blurred by changing the distance of the object, is in no sense helpful but is only harmful for clearing up an image blurred by poor lighting conditions; yet so long as unclear seeing is present, due either to blurring of the image or to unfavorable conditions for its clear discrimination by the retina, the eye will strive by the three adjustments at its command to remedy the deficiency. This striving of the eye to clear up vision by ineffectual maladjustments is the cause of what is commonly called eye-strain and is an important cause of ocular discomfort. The misdirected effort or strain is of no service to vision and leads rapidly to fatigue and exhaustion, to deformities slight in their physical magnitude but great in their functional importance, to inflammations and congestions and to hypertensions and other conditions not found in a healthy eye. Accommodative strain in itself, for example, leads to hyperemia or congestion of the network of blood vessels just in front of and behind the ciliary muscles. The congestion in this so-called danger zone of the eye leads in turn to an increased flow of fluid into the anterior chamber from the blood vessels on the anterior border of the ciliary muscles and into the vitreous chamber from those on the posterior border of these muscles and, perhaps, through the products of the congestion of inflammation, to a blocking up of the drainage angle between the iris and the base of the cornea. This tends to produce a disturbance in the normal balance of the income and outgo of fluids into the interior of the eye and the evils attending such a disturbance.

If the eye could only be educated to be quiet under the poor conditions of seeing for which it has no specific corrective adjustment the cause of vision would be just as well or even better served, and the eye itself would be a great deal better off.

It is somewhat difficult to trace all the effects of eyestrain. It is not the result of normal exercise of the powers of the eye in either kind or amount, and no possible benefit can accrue from it. Rather it leads to an unhealthy condition of the eye, exaggerating and aggravating any abnormal tendencies or predispositions already present, lowering the normal recuperative and restorative powers of the eye and leading sympathetically to functional disturbances in other parts of the body. Like any other organ of the body, the eye if it is to remain healthy or to cure itself of any of its ills, congenital or acquired, must first be put into a situation calling only for the healthy exercise of its normal functions. Important factors in this situation are the conditions under which it is ordinarily called on to work.

OUTSTANDING FEATURES IN LIGHTING IN RELATION TO THE EXAMINATION AND CARE OF THE EYE

The two important aspects of conditions of work as regards the eye are the type of work and the illumination. In relation to the type of work the following are to be considered if the work involves the discrimination of print on paper: the reflecting power of the paper and the type of reflection; the color of the paper; its texture and opacity; the blackness and flatness of the ink used; the size of the page or area of reading space exposed at any one time; the size, shape and spacing of print, and other factors. It is not, however, our purpose to do more than list these items here. In relation to the illumination of the work, we shall consider only four points in the present discussion: the intensity of the illumination, glare, the placement of the light and the color of the light. We have selected these, first, because of their fundamental importance and, second, because we are completely equipped to provide hygienic conditions with respect to them.

Intensity of Illumination.—One of the most outstanding facts with reference to the intensity of the illumination is the wide variation that is found in the amount of light needed and preferred for different types of work, for persons of different ages and for different persons doing the same type of work and in the same age group. Important factors in these individual variations are: age, the refractive condition of the eye, the health of the eye, the clearness of the media, the size of the pupil, susceptibility to glare and the keenness of discrimination of light and space. If too little light is used low visibility results, and the eye undergoes all the strain and malfunctioning of the muscular equipment discussed in the preceding paragraphs. In case too much light is used the excessively harmful effects of glare are experienced. For by far the greater number of persons the range of toleration for the comfortable use of the eyes is comparatively narrow for light of Mazda

quality, and somewhere in this range is a pretty clearly defined preferred intensity. While the correct adjustment of intensity is extremely important in the case of all eyes it is of paramount consideration in the case of those eyes which come under the care of the ophthalmologist. It is not enough to say that one shall or shall not use one's eyes for reading or work or to say that they shall be used only for a certain number of hours a day. The conditions under which they can be used should be prescribed, and the prescription should be accompanied by the information needed to carry out the recommendations. In this way not only would the eyes in question be greatly benefited, but a much greater amount of work could be allowed.

There are two ways of dealing with the situation with regard to the regulation of the intensity of the light to suit individual needs. 1. It can be found what a person's range of toleration of intensity for the comfortable use of the eyes and his preferred intensity are. A prescription can then be given advising what intensity he should have. For this purpose instruments of the type of our variable illuminator¹ can be conveniently used. This procedure is not, however, completely satisfactory, because of the difficulty the patient will have in filling the prescription with such lighting equipment as he is able to procure. In this connection it must be remembered that the preferred intensity must be closely approximated or the prescription will signally fail to accomplish its purpose and, further, that the exact amount of light needed varies with the kind of work, the time of day, the variation in the condition of the eyes and other factors. It is impossible satisfactorily to take account of all these factors in a single test period. Again, the patient may not even attempt to fill the prescription because of the trouble involved and the amount of technical knowledge and understanding it would entail. 2. A much more satisfactory procedure is to use the test only for the purpose of finding out approximately what the patient needs and then to recommend the use of a local lighting unit provided with an intensity control over the range which satisfies his requirement. He can then at any time and for any work adjust the intensity of light exactly to suit his needs. The purpose of the test in this procedure is that the examiner may find out the type of eye he is dealing with for his own enlightenment in relation to the care of the eye and that he may recommend the right type of lighting unit. To meet this need for a variable intensity we have devised desk and table lamps, floor stand lamps and bed reading and examining lamps, provided with mechanical

1. Ferree, C. E., and Rand, G.: Lamp for the Determination and Measurement of the Preferred Intensity of Light for Reading and for Other Work, *Arch. Ophth.* **12**:45 (July) 1934.

controls which change the intensity of the light in continuous series but do not change the color or composition of the light or the size, shape or location of the illuminated area.

It would be an ill considered practice in lighting to make a blanket recommendation of a certain intensity of illumination or a narrow range of intensity for all persons, even for the same kind of work. It will also be realized how difficult it is adequately to handle the problem of intensity by general illumination alone for any considerable number of workers. In some cases it is possible by means of information obtained by testing a group of workers so to distribute them in a room lighted by general illumination as to give some consideration to their individual needs. We have been able to accomplish this result with notable success in a room in a large publishing house used by fifty or more proof-readers. In this case a systematic measurement was made of the intensity of illumination of the room, and each of the proofreaders was tested for the preferred intensity of light and then assigned to a location in the room that most nearly met the requirement. No significant change was made in the lighting of the room. The ideal way to handle the problem, however, would be to provide a suitable intensity of general illumination and use local lighting with variable control of intensity in individual cases.

Glare.—Glare is of two types, simple and veiling. Simple glare is too high brightness in any part of the field of view due to excessive stimulation of the sensorium by light. In the center of the field it is commonly called glare on the work; in the peripheral field it is usually due to the source of light, the lighting fixtures, high reflections and poorly controlled or misplaced intensities of light. Glare on the working surface may also be of the type known as veiling glare. Veiling glare is an obscuring of the image on the retina produced either by an overlay of scattered light or by light reflected from the work surface which is not focused. The former is due to the diffusing or scattering properties of the lens and other media of the eye, and the latter, to specular reflection from the work surface itself. In the latter respect it may be said that the light diffusely reflected from the working surface alone produces an image of that surface on the retina; the light specularly reflected would, if focused, produce an image of the source, not of the working surface. The eye in viewing the working surface naturally adjusts itself to focus the diffusely reflected light. This leaves the light specularly reflected unfocused. In proportion, therefore, as one has specular reflection the image of the working surface is blurred and confused.

The experience of simple glare ranges from a feeling of too much light or an annoying brightness through the various stages of discomfort

to acute pain. Veiling glare by producing confusion in the images causes eyestrain or discomfort through a misdirected and futile effort on the part of the muscles of the eye to clear up its images. That veiling glare causes eyestrain and discomfort is not difficult to understand. It is not so easy to explain the acute pain often caused by simple glare. This is perhaps due to a sharp contraction of the pupil, particularly if the iris is inflamed or supersensitive, or to some condition or reaction set up in the eye itself or in the sensorium. In any event it is a warning or danger sign that harm is being done to the eye. In connection with the pain and discomfort experienced it will be remembered that the iris and other parts of the eye are richly supplied with pain nerves.

Eyes differ greatly in their susceptibility to glare. A study of this susceptibility and of the factors that cause it is an important need in relation to the care and welfare of the eye. It is especially important that susceptibility to glare and the requirements relating to the intensity of light should be studied in the case of diseases of the eye and in the case of some of the more serious defects in refraction and other refractive errors that produce an irritable and supersensitive condition of the eye. We have, for example, observed the acute susceptibility to glare in cases of glaucoma, retinitis pigmentosa, iritis and conjunctivitis, high myopia, marked difference in the refractive condition of the two eyes and uncorrected or poorly corrected astigmatism.

In general the following subjective factors may be noted in connection with susceptibility to glare: age, the size of the pupil, the condition of the media of the eye, the health of the eye and the general health, marked asymmetry of refraction of the two eyes, high myopia and refractive errors that produce an irritable and supersensitive condition of the eye, ocular and bodily fatigue, loss of sleep, insomnia, etc., and the following objective factors may be noted as predisposing to glare: the reflecting power of the surface viewed and the character of the reflection (specular or diffuse), the size of the reflecting surface, the intensity of the light, the angle at which the light falls on the work and the breadth of the angle of the incident light, the diffuseness of the light, the color and composition of the light, high brightnesses in the paracentral and the peripheral field of view and their angular relation to the line of sight, etc.

The most acute problem in modern lighting from the standpoint both of hygiene of the eye and of the designing of lighting equipment has been, it scarcely need be pointed out, the elimination of glare.

Placement of Light and Brightness.—A third important feature in lighting is the correct placement of the light. This problem was created with the devising of lamp-shades for shielding the eye from glare and later giving these shades a reflecting lining to conserve and direct the

light. When the opening of the reflector was turned down the light was directed toward the plane of the work, and the walls and ceiling were left dark or poorly illuminated. On the other hand, when the opening of the reflector was turned up the light was directed to the ceiling and from there reflected to other parts of the room. This resulted in a disproportionately high brightness of the ceiling and a correspondingly low intensity of light on the plane of the work. Relief from the glare of the opening was obtained at the cost of poor and inefficient placement of the light. As a compromise between these extremes in the placement of the light recourse was had to inverted translucent bowls or housings which reflected part of the light to the ceiling and transmitted a part laterally and downward, to opaque housings or reflectors which directed part of the light upward and part downward (direct-indirect units) and to diffusing globes. While these units gave a better placement of light than either the totally direct or the totally indirect units the protection afforded against glare was far from adequate. In a later paper we shall show that the most favorable placement of light and brightness in respect to the walls and the ceiling is that in which the maximum brightness is near the level of the eyes of the worker, with an even and gradual decrease in both directions upward and downward, thus giving the upper part of the walls and the ceiling, for example, less than the maximum brightness. The next step in the development of lighting fixtures has been the use of baffles to give the minimum interference to the distribution of light from the source and the maximum protection against glare. This development has been discussed in former papers² and will be noted later in the present paper.

An important feature in the designing of equipment for both general and local lighting is the provision of a means for varying the placement of light to suit the needs of the particular situation. One of the difficulties in securing good lighting effects at present is the lack of flexibility and variety in this respect. The units that can be had ready-made of a stock or standard type differ widely in the lighting effects that they produce. Thus it is somewhat difficult to adapt the units that are available to such situations as are presented by rooms of different sizes and shapes, of different ceiling heights, characteristics of the walls and the ceiling with respect to contour and surfacing and the purposes for which the room or enclosure is to be used. This difficulty is sometimes met in what is called custom-made lighting by using units of special

2. Ferree, C. E., and Rand, G.: *Lighting Without Glare*, Arch. Ophth. **8**:31 (July) 1932; *Lighting Without Glare: A Further Contribution*, *ibid.* **9**:344 (March) 1933; *The Glareless Lighting of a Dark Room*, Am. J. Psychol. **45**:735 (Oct.) 1933; *A Bed Lamp Designed to Give Glareless Illumination*, Mod. Hosp. **43**:78 (Nov.) 1934.

désign and more than one type of unit in the lighting of the enclosure. A further possibility, as will be noted later in the paper, is to make provision in the unit itself for variability in the placement of the light. This can be done in both the general and the local lighting units, perhaps more easily and conveniently in the local units. It is possible, for example, in the designing of local lighting units practically to eliminate glare from the working surface without undue loss of luminous efficiency by suitable means for varying the direction and placement of the light on the work. A description of this means will be given in a later paper.

Color and Composition of Light.—Among the reasons why the color and the composition of light are to be considered of importance in the care and welfare of the eye the following may be noted: 1. Colored light gives the eye less power than colorless light to see objects of neutral color. In former papers³ we have shown this with light taken from the spectrum, filter colors and colored illuminants. The functions tested were acuity, speed of discrimination, power to sustain clear seeing and ocular fatigue. Black test objects on white backgrounds and the reading page were used as material for the tests. In every case with colorless light, i. e., color-corrected light or light of daylight quality, the subject gave a better performance in all these respects than with colored light. For the spectrum light the best performance was in the yellow part (around the wavelength of 578 millimicrons). On each side of this there was a decrease toward the long and short wavelengths at the ends of the spectrum, the difference in performance varying with the function tested; that is, the decrease was greater for speed of discrimination than for acuity and greater for power to sustain than for speed of discrimination, as might be expected from the fact that the addition of speed and power to sustain adds sensitivity in any performance used as a test. In carrying out these tests the test surface for each color was made of the same brightness and saturation. This equalization as to saturation and brightness was also made in the tests with filter colors and colored illuminants. An important factor in these differences of performance is that the difference in sensation is greater for black on white than for black on any color and greater for black on yellow than for black on any other color. In this connection it will be remembered that the visibility of an object depends on its difference from the background as well as on its size.

3. Ferree, C. E., and Rand, G.: The Effect of Variation of Visual Angle, Intensity and Composition of Light on Important Ocular Functions, *Tr. Illum. Engin. Soc.* **17**:69 (Feb.) 1922; Further Studies of the Effect of Composition of Light on Important Ocular Functions, *ibid.* **19**:424 (May) 1924; Visibility of Objects as Affected by Color and Composition of Light: I. With Lights of Equal Luminosity or Brightness, *Personn. J.* **9**:475 (April) 1931; II. With Lights Equalized in Both Brightness and Saturation, *ibid.* **10**:108 (Aug.) 1931.

2. The color and composition of light, as well as its intensity, are factors in causing glare. This is apparently true for both simple glare and veiling glare. Glare occurs, for example, at lower intensities with artificial light than with daylight and light properly corrected for color. And, again, it seems that colored lights differ among themselves in their tendency to produce glare. Just why these differences should be present in the case of simple glare it is somewhat difficult to understand. In the case of veiling glare they are doubtless due to the difference in the tendency of the media of the eye to scatter lights of different wavelengths and composition.

3. There is a greater tendency for colored light to produce ocular discomfort than for white light to do so. This tendency also differs among the colors themselves. This is found to be true both when the lights in question are used as illuminants and when the eye is exposed to them without any effort or attempt to discriminate detail.

The effect of color on the eye's comfort and performance varies a great deal for different persons. Eyes made irritable and supersensitive by refractive defects and disease seem in general to suffer most from color in light. It may be said, however, that daylight and artificial light properly corrected for color are better for all eyes than colored light.

MEANS FOR IMPROVING LIGHTING CONDITIONS

The lighting in current use is characterized by a high and harmful glare which is becoming worse as the level of the intensity rises, and little means are being provided for adapting the intensity and placement of light to the various needs and conditions that arise. From our point of view, eliminating glare and varying the intensity and the placement of the light as may be needed are three of the four important points in good lighting. The fourth is commercially feasible correction for color.

Work on lighting in relation to the eye is not complete so long as it is confined to the study and investigation of principles and factors. Means must be found for applying what has been learned to the betterment of lighting conditions. After more than twenty years of study and experimentation in relation to factors and principles we have begun to try what we can do to improve lighting conditions, particularly with reference to intensity, glare and placement of light and brightness. We have, for example, devised means whereby any intensity of light that would be needed in both usual and unusual lighting conditions can be secured with complete elimination of glare. This has been accomplished by the use of glare baffles or a plurality of nonreflecting light-shades of suitable size and shape so worked into the design of the fixture or housing of the source of light as to shield the eye completely from the glare of the lamp itself and all auxiliary reflecting surfaces and to give

such wide and uniform distribution of light as to eliminate entirely all high and uneven brightnesses on the walls and ceiling. These baffles have also been used to meet the needs and requirements of local lighting. We have further included in local lighting the important feature of variable intensity of light, as has been noted earlier in the paper, and in both general and local lighting, that of variable placement of light, and provisions have been made for correction of color when that is desired.

Our list of devices includes the following: inset, ceiling and wall-bracket fixtures for commercial lighting and the lighting of hospitals, offices and homes; bed reading and examining lamps; desk and table lamps; floor stand reading and bridge lamps; lamps for the lighting of lecterns and speaker's desks; a device for lighting tunnels and corridors and for protection from glare from automobile headlights, and lamps for the optician's fitting table and the refractionist's chair.

We have considered it worth while to note these devices here in order to show that it is possible to carry out the hygienic measures with reference to lighting that we have recommended for the care and treatment of the eye. Whether the means that we have devised or some other means shall be adopted is of secondary importance. The primary thing is to show that such results can be accomplished in ways that are entirely practical and commercially feasible. A too great obstacle to this end is encountered in relation to those who make and sell lighting equipment. The reasons for this are in part conventional but largely commercial. The change from the traditional form of lighting requires the adoption of new ideas and new ways of thinking, and somewhat radical changes in manufacturing equipment. Further, the manufacturers of lighting equipment contend that there is no convincing public demand for better lighting. If and so far as this is true, there is need for competent and earnest educational service. In this respect the public naturally looks for guidance to physicians, in particular to the eye specialist. Our own experience, however, leads us to believe that the public is willing and eager to have better lighting if they knew what to get and where and how to obtain it.

MEANS AND METHODS OF TESTING

A complete program for prescribing light should have the following features: 1. Tests should be made of the subject's preferred intensity of light and his range of toleration for intensity for the comfortable use of the eyes for the work he is most accustomed to do and for different types of work, of his susceptibility to glare and of his need for correction for color. 2. From the results of the tests just mentioned recommendations should be made as to the intensity of the light that

he should use and the type of lighting equipment that will satisfy his needs in this respect and in respect to protection against glare and harmful coloration of light. With respect to glare, it may be said that, while no eyes should be exposed to glare, more latitude can be allowed to some than to others. It is hoped and expected that in course of time the control of glare in public places at least will become a matter of public health and safety regulation.

All the aforementioned tests can be made easily and conveniently with an instrument that we have called a variable illuminator. The purpose of this instrument is to provide a means of varying the intensity of the illumination in continuous change over a wide range, e. g., from 0 to 100 foot-candles without change in the color or composition of the light or in the size, shape or position of the illuminated area, and to provide a means of correcting artificial light to light of daylight quality. The essential features of the instrument are a source of light of adequate intensity to give the range needed, a housing of suitable size and shape, a mechanical means or specially designed shutter for varying the intensity of the light and a diffusing plate or other diffusing means both to eliminate completely shadows which would otherwise be produced by the shutter and to give an evenly distributed and well diffused illumination of the test surface. The laboratory model of this instrument was described in a former paper¹ and mentioned in a still earlier paper.⁴ It will suffice here to describe the means employed for varying the intensity, for diffusing the light and for giving the correction for color.

The means of varying the intensity consists of four vanes which extend across the opening of the housing in such relation to each other that when their flat surfaces are parallel to the beam of light the maximum amount of light passes through the opening, and when they are rotated to a position at right angles to the beam the light changes in continuous series from full intensity to zero. In changing the intensity the vanes are so activated as to cause the contiguous vanes to turn in opposite directions. This insures absence of shift in the position of the illuminated area and absence of change in its size and shape. When the vanes all move in the same direction, as is the case with Venetian blinds, all these changes take place. In the various models of the instrument different means of activating the vanes have been employed. For one of the models a simple means has been devised which lies within the housing and is entirely free from slack or backlash. This is in the form of a thin plate with a central longitudinal slot through which pass

4. Ferree, C. E.; Rand, G., and Lewis, E. F.: The Effect of Increase of Intensity of Light on the Visual Acuity of Presbyopic and Non-Presbyopic Eyes, *Tr. Illum. Engin. Soc.* **19**:296 (April) 1934.

the axle pins at the ends of the vanes. This slot permits the plate to travel back and forth as the vanes turn. Above and below it in alternate sequence are short vertical slots which engage pins suitably positioned at the ends of the vanes. To the end of the axle of one of the vanes is attached a knob. When this knob is turned the contiguous vanes rotate in opposite directions as is desired.

The intensity of the light on the test surface for any position of the vanes can be read from a calibrated scale suitably positioned on the instrument. The spread of the light from the opening has been checked at several distances to make sure that it follows the law of squares; thus, if a rating of the intensity is desired at any other distance it can readily be computed from the reading on the scale. To secure constancy of current an ammeter and rheostat may be used, if desired. Also the intensity of the light can be measured with a photometer, or light meter, instead of being read from the scale.

In order that no shadow shall be cast by the vanes in any position in their rotation a plate of Belgian flashed opal glass is mounted directly in front of them, between them and the surface to be illuminated. In addition to eliminating completely all shadows on the illuminated surface, this provision secures exceptionally good diffusion of light. For correction for color this diffusion plate may be slipped from its position in front of the vanes and the correcting filter inserted. Any color filter that is desired may be used for this purpose.

The use of such vanes as we have employed in combination with a diffusing plate to eliminate shadows provides a simple mechanical means of varying the intensity of illumination over any range that may be desired and one that is free from the objections as to effects of illumination and inconvenience of use which may be noted in connection with other ways of reducing intensity, such as changing the distance of the source, and the use of a rheostat. Further, this means is readily and easily adaptable to lighting fixtures and equipment of various types and to instruments supplied with special lighting devices.

To meet various needs, models have been designed for mounting on a floor stand, on a bracket which can be clamped on the edge of a table or the arm of a chair and on a stand suitable for desk or table use. In these models the weight has also been greatly reduced, the lightest model weighing approximately 2 pounds (0.9 Kg.). A convenient carrying kit for field work and a suitable handle for holding the unit in any position in relation to the work that is desired have been provided.

Test for the Preferred Intensity of Light.—In earlier papers we have shown the effect of intensity of light on such important func-

tions as acuity and speed of vision⁵ and on acuity at different ages,⁶ and in a paper as yet unpublished we shall show its effect on the speed of vision at different ages. In these papers it was shown in general how much the powers of the eye could be increased by increasing the intensity of the light. However, in such determinations no account is taken of what intensity is preferred for different types of work or what range of intensity is favorable. That is, no account is taken of glare on the work as determining the upper limit, and of diminished acuity as determining the lower limit, of the amount of light that can be used with comfort. The difference between these two limits may be roughly called the range of toleration for the intensity of light. Intensities above the upper limit for comfort may be used in cases of emergency, for example, to give higher visibility, when needed, or a higher speed in the use of the eye, but they can hardly be recommended for ordinary uses of the eye or as best for it over long periods of time.

It is our purpose here to describe a test for the preferred intensity of light and for what we have just called the range of toleration for intensity.

Before making the test the curtains should be drawn and the artificial light in the room turned off. Because of the wide spread of light from the test instrument (140 degrees) general illumination is not needed. The instrument provides a favorable test condition in relation to the illumination of the surrounding field. That is, as the light on the reading page is increased there is a corresponding increase in the amount of light in the surrounding field. This avoids any tendency toward a spot-light effect as the intensity of illumination on the test surface is increased, and gives a type of illumination similar to that which would be obtained in a lighting system in which the increase of intensity is produced without an increase in glare. It should be remembered that there will be a low tolerance for the intensities of light that are needed for good seeing if the increase of intensity is accompanied by an increase in glare. In this connection it may be noted that with our variable illuminator and our glareless local and ceiling lighting units the test can be made and the prescription filled under the same conditions; namely, any intensity of light that may be desired in a practical lighting situation can be obtained without glare.

5. Ferree, C. E., and Rand, G.: The Effect of Variations in Intensity of Illumination on Functions of Importance to the Working Eye, *Tr. Illum. Engin. Soc.* **15**: 769 (Dec.) 1920; The Effect of Variations in Intensity of Illumination on Acuity, Speed of Discrimination, Speed of Accommodation and Other Important Eye Functions, *Tr. Am. Ophth.* **19**:259, 1921; The Effect of Increase of Intensity of Illumination on Acuity and the Question of the Intensity of Illumination of Test Charts, *Am. J. Ophth.* **6**:672 (Aug.) 1923.

6. Ferree, C. E.; Rand, G., and Lewis, E. F.: Age as an Important Factor in the Amount of Light Needed by the Eye, *Arch. Ophth.* **13**:212 (Feb.) 1935.

In conducting a test of this kind no direct light from the instrument should fall on the patient's face to cause objectionable glare. Further, in the case of patients wearing glasses the light should never come from above or behind on either side, because of harmful and annoying reflections from the rims and lenses of the glasses. In making the test with our variable illuminator the patient is seated beside the instrument with his eyes back of the flaring bonnet or shield surrounding the opening of the housing. Thus no light falls directly on his face, and the light does not come from above or behind him. In order to give consistency and correctness of result and avoid the need of making a number of trials, the size of the pupil and the sensitivity of the retina should be standardized by exposing the eye for a suitable length of time to a properly selected intensity of illumination before beginning the test. The omission of this precaution is inexcusably bad technic.

Three sizes of type printed on test sheets of unglazed paper are used, one of the size ordinarily used in printing books (10 point), one of the size used for magazines and newspapers (8 point) and one of the customary fine print used for footnotes (6 point). The examiner may use anything else about which the patient wishes information.

The patient should be seated directly in front of the test material at a distance of about 18 inches (45 cm.) or, in case of a presbyope, at the distance needed for his reading glasses, and his eyes exposed for three minutes to the intensity of illumination on the test surface selected as standard for this purpose. Five foot-candles is suggested because it is judged to be fairly representative of the amount of light that is now being used by the persons who will be tested, the purpose of the test being to find out whether this is sufficient or how much more should be had. Moreover, we have found little difference in the result when the exposure was to 5, 10 and 20 foot-candles and in many cases no difference whatever when the test was made in accordance with the instructions given in the following paragraph, namely, when the series of intensities presented for the patient's judgment is given sufficiently slowly to allow the size of the pupil and the changing sensitivity of the retina adequately to keep pace with the changing amount of light. Except in cases in which the patient's eyes have previously been exposed for some time to very high or very low intensities of illumination we have found three minutes of exposure to be long enough to bring the eyes to a practically constant state of sensitivity to the illumination selected for the preexposure. In this connection it should be remembered that adaptation to light is much more rapid than adaptation to dark. That is, the eye when exposed to light loses sensitivity much faster than it recovers after exposure to light.

The full range of intensity of the instrument should be run to give the patient the experience of the different intensities. Then, beginning below 5 foot-candles, the intensity should be increased very slowly in order to give the size of the pupil and the changing sensitivity of the retina a chance adequately to keep pace with the changing amount of light. The patient should be instructed to report when enough light is had for comfort in reading the test material. This intensity should be recorded and the increase continued very slowly, the patient being asked to report when the intensity is too great for comfort or is beginning to be glaring. The difference between these two points is roughly the favorable range, or the range of toleration for intensity. The patient is next instructed to turn the knob on the intensity control and find for himself just what intensity he most prefers. He should be allowed all the time he desires to make this determination. Two results are thus obtained—the favorable range, or the range of toleration for intensity, and the intensity most preferred. Both these should be recorded on the prescription given the patient. The favorable range, it may be noted, is wider for daylight and for artificial light corrected for color than for uncorrected Mazda light. It is, of course, wider also for eyes insensitive to glare. The tests for determining the amount of light needed for reading should be made for the three sizes of print in order of the largest to the smallest and then should be repeated for the largest size in order to minimize the effect of practice. If there is a difference in the result the second value should be used.

In making a test of this kind the greatest danger that is incurred is that the examiner may force the results or prejudice the mind of the observer as to what intensity he should report as most favorable. It is extremely important that the examiner should himself have no prejudice or allow himself to be prejudiced as to what results should be obtained and what prescription should be given as to the amount of light the patient should use. Correct prescriptions, if followed, will lead, on the whole, to the use of a great deal more light than is now being used, but the light will be distributed in accordance with actual needs. Results of studies made with this test will be given in later papers.

Tests for Susceptibility to Glare.—These tests have the following points of interest: 1. They sustain an important relation to the clinical examination of the eye. A test of simple glare is essentially a test for photophobia, made under better conditions and perhaps with a clearer understanding of the exact purpose of the test than when one is dealing with photophobia. It is not our purpose to give here a clinical evaluation of a test for photophobia, but it may not be out of place to point out that a diagnosis can rarely be made from the results of any one type of

test alone and that it is entirely wrong to gage the value of a test only or even predominantly by its possibilities in this direction. In the selection of tests for a diagnostic program, for example, the important thing is not so much whether one test shows more than another as whether it shows the same thing as that other. All diagnosticians will agree, we think, that competent diagnosis is based on as complete a picture of the condition as it is possible to obtain. Prejudice, inexperience or the lack of a just understanding alone could lead, it would seem, to any other conclusion. A test for veiling glare is a test for clearness or its converse, cloudiness of the media of the eye. Again, this sustains an important relation to more than one pathologic condition of the eye and similarly should be considered as filling out the diagnostic picture. Further, quite aside from questions pertinent to the diagnosis, any test which will serve as a sensitive means of detecting changes in the cloudiness of the media should form a valuable addition to the list of clinical tests. Inspection, it is scarcely needful to point out, is not adequate for this purpose.

2. Tests for susceptibility to glare should have a place in a well rounded and representative program for the general examination of the eye. Like the physical examination in general medicine, such a program sustains a broad relationship the details of which need not be gone into here.

3. Tests for susceptibility to glare have perhaps their greatest importance in relation to the care and treatment of the eye. While all eyes should be protected against glare, great care should be exercised in this respect in the case of eyes which show unusual susceptibility. As will be shown later, the variation in this susceptibility, even among eyes not classed as showing a pathologic condition, is astonishingly great. In relation to tests, proportionately too much stress perhaps is put on their value in regard to diagnosis. Too often after the diagnosis is made one does not know what to do about the condition. In this connection tests which have for their purpose the determination of what is needed for the care and welfare of the eye in question obviously should not be overlooked.

The prime requisite for making a test for susceptibility to glare is to have a means of varying the intensity of the light in continuous series over a sufficiently wide range without altering the color or composition of the light or the size, shape or location of the illuminated area. For this purpose we have two instruments to recommend, our variable illuminator and our instrument for testing the light sense.⁷

7. Ferree, C. E., and Rand, G.: A New Type of Instrument for Testing the Light and Color Sense, *Am. J. Ophth.* **14**:325 (April) 1931.

In the test for simple glare with our variable illuminator, the light may be thrown on a mat white surface of good reflecting power, such as Hering standard white paper, and of suitable size, such as the size of the reading page, this surface to be viewed at the reading distance when the test is being made for susceptibility to glare in reading, or on a sheet of diffusing glassware positioned 5 or 6 inches (12.5 to 15 cm.) in front of the opening of the illuminator. In the latter case the conditions of the test are the same, with the exception that the glare is produced by transmitted light. In using the instrument for testing the light sense for the test, the glare surface is the illuminated field of the instrument. By means of the iris diaphragm provided in the instrument for varying the size of the illuminated field, the glare surface may be adjusted to the size desired. In case a different shape of glare surface is desired, this can be obtained by throwing the iris diaphragm wide open and inserting in the slot provided for the purpose a stop or diaphragm of the shape desired. The test may be conducted in a dark room or, if that is not available, in a room from which all light excepting that coming from the test instrument is excluded as nearly as possible. In order to standardize the sensitivity of the eye, it should be exposed for three minutes to the test field illuminated at some suitable low brightness, which should be kept constant from test to test. The light in the test field should then be varied gradually from low toward high until the threshold of glare or uncomfortable brightness is reached, due precaution being taken with respect to factors that might influence the constancy of this result. Since the size of the pupil is an important factor in susceptibility to glare, an artificial pupil should be used in cases in which it is desired that this factor shall be ruled out. An artificial pupil is provided in the eyepiece of our instrument for testing the light sense. Means for this control are not provided with the variable illuminator. In case this instrument is used the artificial pupil may be worn in a trial frame, or our device for varying the size of the pupil described in an earlier paper⁸ may be used. The latter device is admirable for the purpose.

In making the test for veiling glare also either the variable illuminator or the instrument for testing the light sense may be used. In case the variable illuminator is employed a detail of suitable size and shape differing from the background may be stenciled or impressed at the center of the diffusing plate used as both the background and the surrounding field. The conditions with reference to the amount of light in the room and presensitization or preexposure of the eye may be the same as those described in the test for simple glare. Again the light

8. Ferree, C. E., and Rand, G.: A Convenient Device for Using Artificial Pupils of Different Sizes, *Am. J. Ophth.* **15**:632 (July) 1932.

is varied from low brightness toward high brightness until the detail at the center of the field is completely obscured. Then the intensity should be varied below and above this point until the determination is made with certainty of the highest intensity at which the detail is still visible and the lowest intensity at which it disappears. The average of these two results should give a fairly correct and reproducible value of the threshold of veiling glare. In case the instrument for testing the light sense is used, the detail can be impressed at the center of a plate of clear, colorless glass and this plate can be inserted in the slot provided in front of the field lens. The method of testing is then the same as that described with the variable illuminator.

No attempt should be made to compare the results obtained with the two instruments in testing for either simple glare or veiling glare. Either instrument should give consistent results from time to time with the same person and comparable results for various persons and various places, but one instrument cannot be substituted for the other. In the latter connection it should be borne in mind with reference to the determination of simple glare that the test field in the two instruments is obtained in different ways. With the variable illuminator it is obtained by illuminating a diffusely reflecting or diffusely transmitting surface, and this is viewed by the eye in the ordinary way, while with the instrument for testing the light sense it is obtained by focusing a collimated beam of light in the pupil of the eye by means of a lens, on the principle that when a parallel beam is focused in the pupil of the eye the lens is seen as uniformly filled with light. In the case of the test for veiling glare, not only this difference is to be considered but the fact that there is presented in the variable illuminator an initial amount of scatter of light due to the diffusing action of the test surface, while in the instrument for testing the light sense there is present only the scattering action of the media of the eye. Just how much this initial scatter, by multiplying the number of angles at which the light enters the eye, influences the scatter caused by the media of the eye and thereby perhaps influences the sensitivity of the test cannot be told at this time.

In the test just described, it will be noted, the visibility of the detail to be discriminated is held constant and the intensity of the obscuring or glare-producing light is changed until the threshold of discrimination is obtained. It is obvious that the converse of this principle could also be used; namely, the intensity of the obscuring or glare-producing light could be kept constant, and the visibility of the detail to be discriminated could be varied until the threshold of visibility was obtained. If the latter principle were used our variable illuminator or some device provided with a shutter means of changing the intensity of illumination could be employed with convenience and advantage to vary the visibility of the detail to be discriminated.

The practical test, considered in relation to prescribing light, is a composite one which takes into account both simple glare and veiling glare. We refer to the determination of the upper limit of intensity for comfortable reading, which has been described under the heading "Test for the Preferred Intensity of Light." This test has the further advantage that it is made in the routine procedure of testing for the preferred intensity of light. It will be understood, of course, that in nonpathologic conditions simple glare is the major factor in this determination.

We have studied with this test five hundred and fifty subjects with no pathologic condition of the eyes, ranging in age from 10 to 77 years.⁹ A brief summary of the results obtained may be given here to show the wide range of variability that may be expected in susceptibility to glare in healthy eyes as measured by the upper limit of intensity for the comfortable reading of a page of ordinary type (8 point) and the importance of the need for testing for this susceptibility in relation to the care of the eye. An upper limit of intensity for comfortable reading of less than 5 foot-candles was found in 1.7 per cent of the cases. This limit was between 5 and 10 foot-candles in 11.3 per cent, between 10 and 15 foot-candles in 26.5 per cent, between 15 and 20 foot-candles in 20.8 per cent, between 20 and 25 foot-candles in 11.2 per cent, between 25 and 30 foot-candles in 6.5 per cent, between 30 and 35 foot-candles in 4.7 per cent, between 35 and 40 foot-candles in 3.3 per cent, between 40 and 45 foot-candles in 2.2 per cent and between 45 and 50 foot-candles in 3 per cent. The remaining 8.8 per cent had an upper limit of toleration for intensity of between 50 and 100 foot-candles. Thus it will be seen that for 13 per cent the upper limit of intensity for comfortable reading was less than 10 foot-candles; for 14 per cent, above 40 foot-candles; for 73 per cent, between 10 and 40 foot-candles; for 65 per cent, between 10 and 30 foot-candles; for 58.5 per cent, between 10 and 25 foot-candles, and for 47.3 per cent, between 10 and 20 foot-candles. From these data it might be considered that those who have an upper limit of less than 10 foot-candles are more than usually sensitive to glare and that those who have an upper limit of less than 5 foot-candles are extremely sensitive to glare, while those who have an upper limit of more than 30 or 40 foot-candles are less than normally sensitive to glare.

Test for the Need of Correction for Color.—The purpose of such tests is a determination of a condition which might be called chromophobia or chromasthenopia, that is, an unusual susceptibility to discomfort due to color in light. In the case of all eyes there is, so far as we know, a preference for light of daylight color. Reasons for this are

9. This study was made in collaboration with E. F. Lewis.

the greater comfort and efficiency that it gives, a lesser tendency to produce both simple glare and veiling glare and the higher visibility that is obtained for objects and backgrounds of neutral color, such as black objects on white backgrounds. There seems, however, to be no pressing reason for using the test unless the history shows an undue amount of physiologic aversion to artificial light, although many are more than willing to bear the expense of correction for color for the sake of the additional comfort and benefit it gives. The particular need for it is in those cases in which there is a complaint of distress in working under artificial light. In such cases it should be used in conjunction with tests for preferred intensity of light and susceptibility to glare in the endeavor to isolate the cause of the trouble. It would be well for the physician to take advantage of the information that can be had from these tests in all cases in which he is accustomed to proscribe entirely or to limit the use of artificial light. In our own experience we have had a great deal of success in prescribing the use of color-corrected light in cases of diseases of the eye and of troublesome refractive defects or in cases in which there is undue nervous and ocular irritability.

Space might perhaps be allowed here for two examples. One is that of a distinguished research worker, scholar and teacher about 70 years of age, the acknowledged leader in his field. This patient was entirely unable to work at night under ordinary conditions of lighting. So far as was known, there were in his case no pathologic condition of the eyes and no unusual refractive defect. Under artificial light properly corrected for color he was, according to his own statement, able to work, if he chose, until 2 or 3 a. m. His own version of the result was that color-corrected light had saved his nights for him. The other case is that of a well known editor and journalist about 60 years of age. This patient had lost the sight of one eye owing to disease, and the condition of the other eye was poor. He had been forbidden to work or read at all under artificial light. His testimony likewise was that color-corrected light made it possible for him to work at night. So pleased was he with the result that he had color-corrected light installed all through his house and was, the last time that we talked with him, contemplating having it installed in his printing plant, stating that what had given him so great a benefit would, he thought, be good for his employees.

Complete correction for color involves a heavy loss of light. It is well, therefore, for the examiner to be provided with filters giving different amounts of correction for color in order to meet the requirements of different eyes and different lighting conditions. Filters suggested are the Daylight and Whiterlite glasses furnished by the Macbeth Day-

lighting Company, etched to give the required amount of diffusion. The Daylight glass used as a corrective of Mazda light gives a color approximating that of an overcast sky. In the scale of color temperatures this may be specified as about 6,500 K. (Kelvin). This color temperature has been adopted by the Bureau of Standards as best for work with color. The color temperature of Mazda light filtered through the Whiterlite filter may be specified as around 5,500 K., depending on the wattage of the lamp. Some benefit may be had by the use of the Celestialite glassware furnished by the Gleason-Tiebout Glass Company. Filtered through this glass Mazda light is relieved of a great deal of the yellow quality that is so distressing to some eyes and to some extent objectionable to all eyes. This glass gives a high transmission of light which makes it feasible for use in all commercial lighting. A further advantage is its high coefficient of diffusion. This glass may be added, if desired, to the test filters already recommended.

In selecting the color filter great care should be taken that the filtered light obtained has a proportion of wavelengths well balanced with respect to the welfare of the eye, such as is the case with the two types of glass supplied by the Macbeth Daylighting Company. Not every blue glass can be used to advantage as a filter for Mazda light. On examination with the spectrophotometer the blue bulb of commerce, for example, shows an excess of light in the green part of the spectrum. Also, the glass used in making this bulb is not carefully standardized; that is, it is variable in its filtering properties. Tests made by us for ocular fatigue and discomfort with this lamp gave a poorer result than that of tests made with unfiltered Mazda light.

For one who really needs light-prescribing for color little more is required in the way of a test than to give him a chance to read or work under color-corrected light. For him the benefit of color correction is so great that there is no doubt as to the preference. However, if a more exacting test is desired, the test for the preferred intensity and the range of toleration of intensity can be made for both corrected and uncorrected light. Thus the range of intensities used affords the observer ample opportunity to judge which gives the better results. Or the comparison can be made at equal intensities, for example, the preferred intensity for the uncorrected light or some other representative intensity. The test should be made with all light as nearly as possible excluded from the room except that which comes from the test instrument.

As a final comment on the merits of color-corrected light we may mention some results that we have recently obtained with an instrument which we devised in 1932. We have called this instrument a visibility comparator. Measurements made with it showed in general that given visibilities in Mazda light could be equaled by the use of a much lower

intensity of artificial daylight, the amount of this difference varying for a given observer with such factors as the level of the intensity used and the size of the print or other detail. In the current practice of lighting the tendency has been to try to increase visibility by increasing the intensity of the light alone. In this practice a serious difficulty is encountered in the glare and ocular discomfort that are apt to be caused by the use of these higher intensities. In the case of many eyes this difficulty is serious and often arises in those cases in which increase of visibility is most needed. In this connection the great effectiveness of color-corrected light for increasing visibility should not be overlooked. In fact, the best conditions for clear and comfortable seeing must include an optimum combination of intensity of light, color of light and size of detail. We particularly feel the need of stressing the importance of the factor of correctness of color. From the standpoint of the welfare of the eye there is everything to be said for this factor and nothing against it.

In case the examiner should want to test the preference of the patient with reference to any of the tinted lenses that are sometimes prescribed either or both of the aforementioned procedures should serve this purpose well. Particularly the test for the preferred intensity and the range of toleration for intensity with both the tinted lens and the neutral lens should give as good an opportunity for making the comparison as can readily be had in a test procedure. In the case of tinted lenses it should perhaps be expected that the benefit, when present, would be largely in terms of comfort rather than visibility. Quantitative tests for visibility are therefore apt to be disappointing. The test can be made, of course, with both artificial light and color-corrected light. It would be expected, however, that the need for the test would be greater with artificial light.

INTENSITY OF LIGHT AND STRENGTH OF READING GLASSES

In any series of tests which is carried out as a basis for the prescribing of the intensity of light it will be found that the major need for individual testing is for the adolescent, the sick and the defective and for persons with presbyopic or with near-presbyopic eyes. There is much less need for individual testing of normal eyes of persons between the ages of 20 and 35 years. We have found that the greater number of these eyes prefer a medium intensity of light over a comparatively small range. In case of the presbyopic eye the greatest benefit from the test may be had for eyes in the early and middle stages of presbyopia. These comments are based on the results of the study of eyes of persons of different ages, noted earlier in the paper.

There are two means of aiding the presbyopic eye to see an object at the desired near distance, namely, a correcting glass and intensity of light. The former of these is the major aid, and the latter, a minor or auxiliary aid. However, the proper selection of either cannot be made without reference to the other. The preferred procedure is the selection of the optimum combination of intensity of light and strength of glass.

So far as the correcting glass is concerned it may be said that the strength of the glass required at a given near distance sustains, within comparatively narrow but significant limits, an inverse relation to the intensity of the light used. At high intensities the work can be discriminated with a weaker glass than at medium and low intensities. This is due for the greater part, if not entirely, to the effect of the higher intensities of light on acuity and the visibility of the object. At medium and low intensities stronger glasses are needed. Through this range, however, there is less effect of change of intensity on the preferred strength of glass—little, in fact, until very low illuminations are reached. At these intensities the tendency is to choose a still stronger glass in order to secure a benefit from magnification. This glass, however, would not in most cases be satisfactory at medium and high intensities without changing the distance of the work, and probably not even then, because of the greater amount of convergence required, the less favorable relation between accommodation and convergence and the undue limitation of the range of distance over which the object can be discriminated. At these intensities the additional magnification is not needed for the comfortable discrimination of the object.

It cannot be too strongly urged that due attention be given to both the intensity of the light and the strength of the glass in prescribing the conditions of seeing for the presbyopic eye. Often too strong glasses are prescribed because too low an intensity of light has been used in making the examination; and, conversely, frequently too high an intensity of light is advised or required because the person is wearing too weak a glass, in which case he may be compelled for the sake of adequate visibility to work above his threshold of glare. A fuller discussion of this topic with illustrative data has been given in a previous paper.¹⁰ Only enough is given here to indicate its relation to the general subject.

SUMMARY

In comparison with other branches of medicine the development and use of hygienic measures in the care and treatment of the eye seem to have suffered retardation, perhaps unduly so. The subject of hygiene has been built up in the attempt to preserve and restore normality

10. Ferree, C. E., and Rand, G.: The Use of Variable Illumination in the Correction of the Presbyopic Eye, *Am. J. Ophth.* 19:238 (March) 1936.

of function through the regulation of the conditions under which the organ functions. Like any other organ of the body, the eye, if it is to remain healthy or to cure itself of any of its ills, congenital or acquired, must first be put into a situation calling only for the healthy exercise of its normal functions. Important factors in this situation are the conditions under which it is ordinarily called on to work, two important aspects of which are the type of work and its illumination. Of these aspects the latter is the more amenable to variation and control. Radical changes cannot be made in the work itself, although some improvement in this direction may come with time. Obviously the lighting of the work is the more promising and the more important angle of attack.

For more than twenty years we have studied lighting in relation to the eye. The results obtained and the principles discovered, however, belong essentially to the general subject of lighting. They are not sufficiently specific to be of direct and significant service to the physician. For him the subject of lighting narrows down primarily to what is needed for the individual patient who comes under his care. For the determination of this special tests which can be easily and conveniently made are needed. The next step is to see that what is needed can be obtained; in short, the prescription must be filled. In later years we have devoted a great deal of our work in lighting to those ends. Means and methods of testing have been devised which cover the more important aspects of lighting, and with them we have studied the characteristics and variability of individual needs in both nonpathologic and pathologic conditions. Lighting equipment of sufficient range and flexibility to meet individual requirements has also been devised, and a great deal of time has been spent in making it available to the public. Thus a new division of the subject of lighting has been created which belongs in its narrower and more technical aspects to the medical profession and in its broader and more general aspects to the welfare and personnel worker. The prescribing of light to meet the requirements of the individual person has become a practical possibility, and we strongly believe that when properly used it will be of significant service in the care and treatment of the eye. The need for it is acute in the case of many of the patients who come under the physician's care.

GROWTH IN MASS AND VOLUME OF THE HUMAN LENS IN POSTNATAL LIFE

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AND

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MINNEAPOLIS

The postnatal growth in mass and volume of the human lens is different from that of most structures associated with the nervous system. Examination of the available published data has yielded records of the weights and volumes of approximately 230 fresh and presumably normal human lenses, and we have been able to supplement these with records of the weight of the lens in ten full term new-born children.¹

These data are sufficient to permit tracing a curve for the postnatal growth of the lens and the computation of certain constants. They are not sufficiently extensive to warrant analytic treatment of the records for subjects in the first decade.

GROWTH OF THE LENS IN WEIGHT

Tables 1, 2 and 3 and figures 1 and 2 illustrate the growth of the lens in mass. The lens has a mean weight of almost 66 mg. at birth. This weight is increased about one-half in the first six months, almost

From the Graduate School and the Institute of Child Welfare, the University of Minnesota.

1. Becker, O.: *Zur Anatomie der gesunden und kranken Linse*, Wiesbaden, J. F. Bergmann, 1883. Clapp, C. A.: *A Communication on Infant Lenses and Their Solids*, Arch. Ophth. **42**:618, 1913. Collins, W. J.: *Lectures on the Anatomy and Pathology of the Eye*, Lancet **2**:1329, 1894; *An Address on the Crystalline Lens in Health and in Cataract*, Brit. M. J. **2**:1440, 1905. Deutschmann, R.: *Fortgesetzte Untersuchungen zur Pathogenese der Cataracte: II. Cataracta senilis*, Arch. f. Ophth. **25**:213, 1879. Engel, J.: *Ein Beitrag zur Physik des Auges*, Vrtljschr. f. prakt. Heilk. **7**:167, 1850. Heine, L.: *Beiträge zur Physiologie und Pathologie der Linse*, Arch. f. Ophth. **46**:525, 1898. von Jaeger, E.: *Ueber die Einstellung des dioptrischen Apparates im menschlichen Auge*, Vienna, L. W. Seidel & Sohn, 1861. Smith, P.: *The Growth of the Crystalline Lens*, Brit. M. J. **1**:112, 1883; *On the Growth of the Crystalline Lens*, Tr. Ophth. Soc. U. Kingdom **3**:79, 1883; *On the Pathology and Treatment of Glaucoma*, London, J. & A. Churchill, 1891. The studies of von Jaeger and others have shown how important it is to determine the weight of the lens as soon as possible after death. The lens begins to absorb water from the surrounding tissues within a short time post mortem. An attempt has been made to eliminate weights that were taken after this postmortem change became effective.

doubled in the first year after birth and increased more than twofold in the early part of the first decade. Thereafter the scheme of growth seems to undergo a marked change, and the increase in the weight of the lens is roughly proportional to age until late senility. At 35 years of age the weight of the lens is approximately three times the weight at birth, and at 75 years it approaches four times the weight at birth.

TABLE 1.—*Weight and Volume of the Human Lens in Postnatal Life**

Age		Weight of Lens			Volume of Lens		
		Number of Lenses	Mean and Probable Error, Mg.	Coefficient of Variation, Percentage	Number of Lenses	Mean and Probable Error, Cu.Mm.	Coefficient of Variation, Percentage
Range	Median						
Birth	10	65.6 ± 1.9	13.7
1 to 3 months	2 months	24	92.8 ± 1.2	9.6
4 to 5 months	4.5 months	4	109.0 ± 6.1	16.6
10 to 11 months	10.5 months	2	124.5
1 to 10 years	3 years	1	146.8
10 to 20 years	17.5 years	6	152.8 ± 2.1	9.0
20 to 30 years	25 years	24	172.0 ± 2.0	9.7	21	162.9 ± 1.8	7.2
30 to 40 years	35 years	31	190.3 ± 1.5	6.4	22	177.3 ± 1.7	6.5
40 to 50 years	45 years	34	202.4 ± 1.9	8.1	23	188.1 ± 2.1	7.9
50 to 60 years	55 years	25	222.3 ± 2.5	8.2	22	205.4 ± 2.7	8.8
60 to 70 years	65 years	41	230.1 ± 3.1	12.8	32	213.0 ± 3.0	11.8
70 to 80 years	75 years	22	237.1 ± 3.4	9.8	21	218.3 ± 2.9	8.8
80 to 90 years	85 years	15	258.1 ± 2.8	6.2	15	238.7 ± 3.0	7.0

* All the values were calculated to the third and thrown to the first decimal.

TABLE 2.—*Observed and Calculated Weight of the Human Lens at Ages from 10 to 90 Years**

Age, Years		Observed (a), Mg.	Calculated (b), Mg.	Residuals†		Calculated at Decades	
				Absolute (a - b), Mg.	Relative ($\frac{a-b}{a} \times 100$), Percentage	Age, Years	Weight of Lens, Mg.
Range	Median						
10 to 20	17.5	152.8	161.4	-8.6	-5.6	10‡	151.7
20 to 30	25	172.0	172.8	-0.8	-0.5	20	166.0
30 to 40	35	190.3	187.1	+3.2	+1.7	30	180.3
40 to 50	45	202.4	201.4	+1.0	+0.5	40	194.6
50 to 60	55	222.3	215.7	+6.6	+3.0	50	208.9
60 to 70	65	230.1	230.0	+0.1	+0.0	60	223.2
70 to 80	75	237.1	244.3	-7.2	-3.0	70	237.5
80 to 90	85	258.1	258.6	-0.5	-0.2	80	251.8
						90‡	266.1

* Empirically expressed, the weight of the lens equals 137.45 mg. plus 1.429 years (age).

† The mean unweighted absolute residual of calculated from observed values, taken without regard to sign, was 3.5 mg. The mean unweighted relative residual of calculated from observed values, taken without regard to sign, was 1.8 per cent.

‡ Extrapolated.

The data that we have collated show sufficient constancy in trend to warrant their expression by an empirical formula for a straight line:

$$LW = 137.115 \text{ mg.} + 1.429 A,$$

in which LW is the weight of the lens in milligrams and A is the age in years. This expression was obtained by fitting, by the method of

averages, the mean weight for each decade at the median point of the decade. The mean values were weighted by the square root of the number of observations included. The calculated figures show an unusually close approximation to the observed values for biologic data of this nature. Table 2 and the upper curve in figure 2 present the results in detail.

The variability of the material is indicated by the coefficient of variation (shown in the fifth column of table 1). This is irregular and is higher than that found for most measurements of the structures asso-

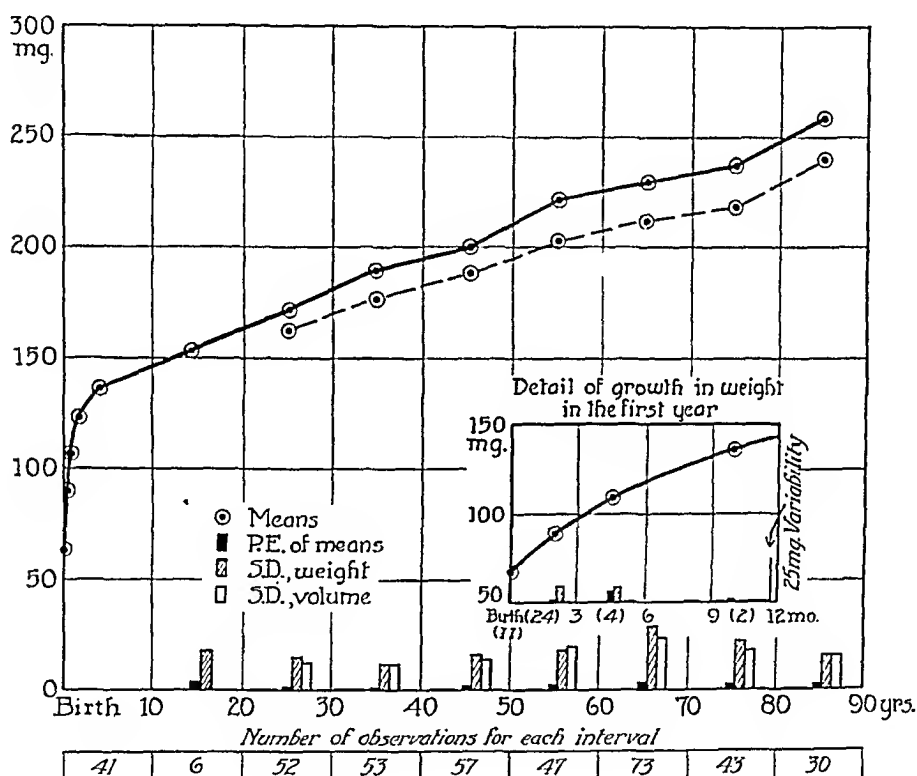


Fig. 1.—Curves showing the growth of the lens in weight from birth (unbroken line) to 90 years and the growth in volume in cubic millimeters (broken line) from 20 to 90 years. The curve for the weight of the lens in the first decade was drawn from inspection. The curves for the weight of the lens and the volume of the lens from 25 years up were drawn by connecting mean points. The inset shows the growth of the lens in weight in the first year; this curve was drawn from inspection. P. E. indicates probable error, and S. D., standard deviation.

ciated with the nervous system but is somewhat below that for most other organs and parts of the body. It seems probable that this apparent variability is increased by the length of the intervals employed and perhaps by experimental error in weighing the lenses.

GROWTH OF LENS IN VOLUME

We have found no reliable data on the growth of the lens in volume in the first two decades, but we have collated 186 observations on the volume of older and presumably normal lenses.² These are shown in the last two columns of table 1 and in table 3 and are presented graphically in figures 1 and 2.

The volume of the lens in persons from 20 to 90 years of age increases at a steady rate which, like the weight of the lens, is proportional to age. In the middle of the third decade the mean volume of the lens is, roughly, 163 cu. mm., and this volume is increased about one third by the time extreme old age is reached.

TABLE 3.—*Observed and Calculated Volume of the Human Lens at Ages from 20 to 90 Years**

Age, Years		Observed (a), Cu.Mm.	Calculated (b), Cu.Mm.	Residuals†		Calculated at Decades	
				Absolute (a - b), Cu.Mm.	Relative $\frac{a-b}{a} \times 100$, Percentage	Age, Years	Volume of Lens, Cu.Mm.
Range	Median						
20 to 30	25	162.9	166.3	-3.4	-2.1	20‡	160.6
30 to 40	35	177.3	177.7	-0.4	-0.2	30	172.0
40 to 50	45	183.1	189.1	-1.0	-0.5	40	183.4
50 to 60	55	205.4	200.5	+4.9	+2.4	50	191.8
60 to 70	65	213.0	211.9	+1.1	+0.5	60	206.2
70 to 80	75	218.3	223.3	-5.0	-2.3	70	217.6
80 to 90	85	238.7	234.7	+4.0	+1.7	80	229.0
						90‡	239.0

* Empirically expressed, the volume of the lens equals 137.8 eu.mm. plus 1.14 years (age).

† The mean unweighted absolute residual of calculated from observed values, taken without regard to sign, was 2.8 eu.mm. The mean unweighted relative residual of calculated from observed values, taken without regard to sign, was 1.4 per cent.

‡ Extrapolated.

As in the case of the weight of the lens, the relation between the volume of the lens and age may be expressed by a simple empirical formula representing a straight line:

$$LV = 137.80 \text{ cu. mm.} + 1.14 A,$$

in which LV is the volume of the lens in cubic millimeters, and A is the age in years. The fit of this expression to the observed means is slightly better than the fit of the expression for the weight of the lens.

RELATION BETWEEN WEIGHT AND VOLUME OF THE LENS

A number of determinations of the specific gravity of the lens have been made, but since these were usually taken without controlled technic, were based on few observations and were without data on age,

2. Most of the data are from the sources cited in footnote 1.

the figures are variable. Thus, Davy³ reported a mean value of 1.100. Nunneley⁴ noted a value of 1.1304 for the lens of a young man, and values of 1.10909 and 1.10967, respectively, for the right and the left lens of an older woman. Chenevix⁵ obtained a value of 1.079. We have computed the specific gravity for the lens from 20 years of age up from our calculated values as given in tables 2 and 3. Such a computation is not entirely trustworthy, for it is based on values arrived at by calculation, and the original values which form the basis of this procedure are averages. The resulting expression therefore approaches

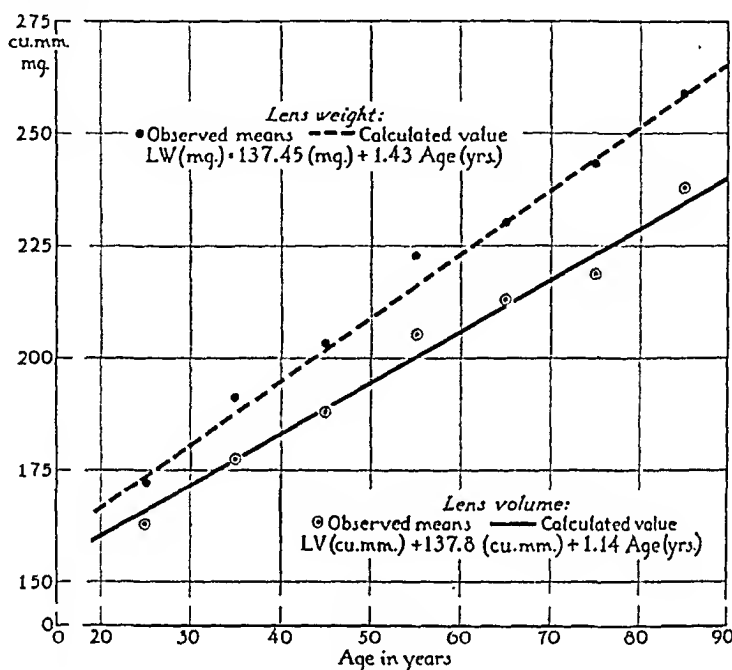


Fig. 2.—Growth of the lens in weight and volume from 20 to 90 years. The broken line indicates the calculated weight of the lens; the solid line, the calculated volume of the lens; the solid dots, the means (for decades) of the weight of the lens, and the circled dots, the means (for decades) of the volume of the lens. These schemata are based on the material given in tables 1, 2 and 3.

the mean of the sums of the ratios and not the average of the individual ratios. The expression is as follows:

$$Sp. Gr. = \frac{LW}{LV} \text{ or } \frac{a + bA}{c + dA}$$

in which *Sp. Gr.* is specific gravity, *LW* and *LV* are the weight of the lens and the volume of the lens, respectively, *A* is the age in years, and

3. Davy, J.: Tr. Med.-Chir. Soc. Edinburgh 3:436, 1829.

4. Nunneley, T.: On the Form, Density and Structure of the Crystalline Lens, Quart. J. Micr. Sc. 6:136, 1858.

5. Chenevix, quoted by Sappey, P. C.: Traité d'anatomie descriptive, ed. 3, Paris, V.-A. Delahaye, 1877, vol. 3, p. 807.

a , b , c and d are the constants given in the first and second formulas. The rate and relative rate of change in this index have been computed by differentiating and are shown in table 4 and figure 3.

TABLE 4.—*Calculated Specific Gravity of the Human Lens at Ages from 20 to 90 Years*

Age, Years	Calculated Specific Gravity*	Rate of Change in Calculated Specific Gravity	
		Absolute, Units per Decade	Relative, Percentage per Decade
20.....	1034	15.6	1.5
30.....	1048	13.6	1.3
40.....	1061	12.0	1.1
50.....	1072	10.6	1.0
60.....	1082	9.5	0.9
70.....	1091	8.5	0.8
80.....	1100	7.7	0.7
90.....	1113	7.0	0.6

* The usual specific gravity, which is a ratio, has been multiplied by 1,000, as is often done in physiologic studies, to avoid decimals.

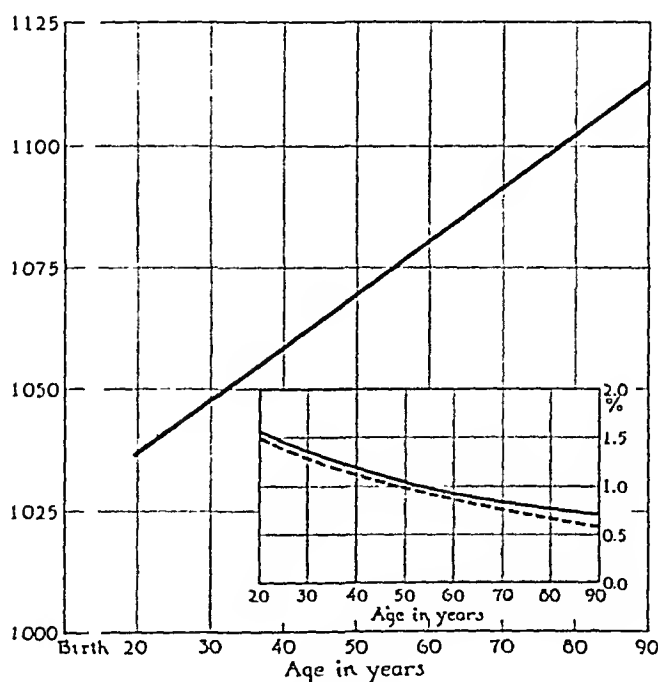


Fig. 3.—The curve in the main panel shows the computed specific gravity of the human lens from 20 to 90 years, inclusive. The curves in the minor panel show the absolute and the relative changes in the computed specific gravity of the lens. The solid curve indicates the rate of change per decade in terms of units; the broken curve, the change per decade in terms of percentage. The specific gravity has been multiplied by 1,000 to eliminate the use of decimals.

The calculated specific gravity is 1034. at 20 years of age, 1072 at 50 years and 1113 at 90 years. The absolute increase as well as the relative increase in specific gravity is much more rapid in the earlier years than in the later years, contrary to common opinion.

It is evident that while the weight of the lens in later years is increased somewhat, owing to its greater density, most of the change is due to the increase in bulk.

COMPARISON OF THE GROWTH OF THE LENS WITH THAT OF
OTHER STRUCTURES OF OR ASSOCIATED WITH
THE NERVOUS SYSTEM

It appears that the weight and probably the volume of the lens show a curious pattern of growth. In infancy and early childhood the growth follows the pattern common to most parts of both the central and the peripheral nervous system. But thereafter growth is continued at a steady rate, whereas most structures associated with the nervous system approach or reach their definitive size in the second decade.

TABLE 5.—*Ratios of Size of Sundry Structures Associated with the Nervous System at Approximately 3 Years and Approximately 20 Years of Age to Their Size at Birth*

Part	Ratio, Percentage		Comment
	About 3 Years	About 20 Years	
Encephalon (weight).....	316	388	Calculated
Cerebrum (weight).....	297	377	Calculated
Cerebellum (weight).....	562	711	Calculated
Brain stem (weight).....	350	526	Calculated
Spinal cord (weight).....	440	900	Estimated
Peripheral nerves of upper extremity (weight)	403	1,530	Data of Heptner (Arch. f. Anat., 1915, p. 277)*
Internal ear (volume).....	100	100	Determined from casts
Optic nerve (volume).....	...	213	Computed from linear measurements†
Combined coats of the eyeball (volume)	...	700	Calculations of Seammon and Armstrong (J. Comp. Neurol. 38: 165, 1925)
Eyeball in toto (weight).....	118	468	Derived from values on smoothed curve
Lens (weight).....	222	253	Calculated

* The great relative postnatal increase in the size of the peripheral nerves is probably due in large measure to the growth of connective tissue and the deposition of fat.

† Seammon, R. E., and Armstrong, E. L.: J. Comp. Neurol. 38: 165, 1925; Ottersky, E.: Untersuchungen über Weichteile und Knochen der mittleren Schädelgrube insbesondere über die Lage des Chiasma opticum, Königsberg, R. Sebenk, 1896.

The amount of postnatal growth of the lens (computed as a percentage ratio to the weight at birth) is also different from that of most other parts of the nervous system. This is illustrated in table 5, which gives the ratios of the size of a number of structures associated with the nervous system at about the age of 3 years and about the age of 20 years to the size at birth. While it is not thought that the ratios given in this table are entirely accurate, they seem to be substantially correct.

It will be seen that most parts associated with the nervous system have made a greater relative gain in the first three years of life than

the lens, although this is not true of the eyeball as a whole. At the age of 20 years all the structures listed, aside from the optic nerve and the internal ear, have made a larger relative gain than the lens. The optic parts as represented by the combined coats of the eye and the eyeball in toto show the same characteristics as the other structures associated with the nervous system. In the first two decades the lens is apparently a decreasing component of the eyeball. This is shown graphically in figure 4, which shows median sagittal sections of the eyeball with the lens, at 5 fetal (lunar) months, birth and early maturity, respectively. In order to make these schemata geometrically correct, the drawings have been reduced to the same area in each instance. After early maturity the lens must necessarily become an increasingly larger component of the eyeball, for all the present evidence indicates that the former structure continues to grow, while the latter remains stationary.

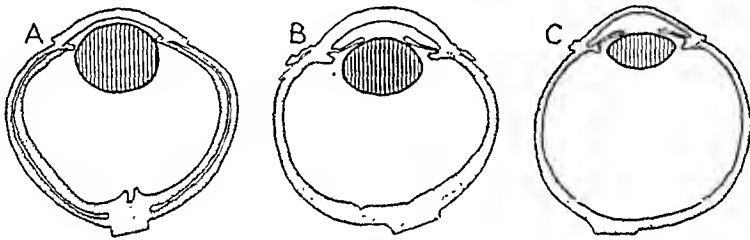


Fig. 4.—Outlines of median sagittal sections of the eye at three ages. *A*, section of the eye of a fetus of 5 lunar months (from Bach, L., and Seefelder, R.: *Atlas zur Entwicklungsgeschichte des menschlichen Auges*, Leipzig, W. Engelmann, 1914). *B*, section of the eye of a new-born infant (from Merkel, F., and Orr, A. W.: *Das Auge des Neugeborenen an einem schematischen Durchschnitt erläutert*, *Anat. Hefte* 1:271, 1892). *C*, section of the eye of a young adult (from Merkel, F., and Kallius, E.: *Makroskopische Anatomie*, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, W. Engelmann, 1892, vol. 1, pt. 1, chap. 1). All the drawings have been reduced to equal areas to establish proportional dimensions.

COMPARISON OF THE GROWTH OF THE LENS WITH THAT OF OTHER STRUCTURES OF THE BODY EXCLU- SIVE OF THE NERVOUS SYSTEM

Not only do structures associated with the nervous system cease to grow at a relatively early age, but also most other parts of the body make little gain or often regress in size after middle life. But it may be pointed out that at least two other structures also increase in size to extreme old age. These are the greater arteries, as determined by measurements either of the caliber or of the circumference, and the alveoli of the lungs, as determined by measurements of the diameter.

A considerable amount of data on the increase in caliber of the great arteries was collected by Beneke,⁶ Thoma,⁷ Suter,⁸ Husten⁹ and others, who all obtained substantially the same results.

The data on the diameter of the alveoli of the lungs were given in a thesis by Rossignol,¹⁰ who found a continued increase in size from early infancy to senility. While the material was collected many years ago, it seems authentic.

It may be suggested that function in each of these organs is associated with elasticity, and that their increase in size, whether it is regarded as physiologic or pathologic, may be associated with the loss of this functional elasticity.

SUMMARY

The postnatal growth in mass and volume of the human lens shows two distinct phases. In infancy and early childhood the lens grows much like a number of other structures associated with the nervous system. After this early phase of rapid relative increment, the lens enters on a period of slow and steady growth, which (as evidenced by specimens selected by the best available criteria of normality) continues throughout the remainder of life. There seems to be no good evidence that increase in density is the major factor in this later growth in mass. It is suggested that the continued growth of the lens through middle age and old age may be associated with loss of elasticity in this structure.

6. Beneke, F. W.: Die anatomischen Grundlagen der Constitutionsanomalien des Menschen, Marburg, N. G. Elwert, 1878.

7. Thoma, R.: Untersuchungen über die Grösse und das Gewicht der anatomischen Bestandtheile des menschlichen Körpers im gesunden und im kranken Zustande, Leipzig, F. C. W. Vogel, 1882; Untersuchungen über die Histogenese und Histomechanik des Gefäss-systems, Stuttgart, F. Enke, 1893.

8. Suter, F.: Ueber das Verhalten des Aortenumfangs unter physiologischen und pathologischen Bedingungen, Arch. f. exper. Path. u. Pharmacol. **39**:289, 1897.

9. Husten, K.: Anatomische und histologische Untersuchungen über Weite und Wand der Hohlvenen, Veröffentl. a. d. Kriegs- u. Konstitutionspath. **16**:1, 1926.

10. Rossignol: Recherches sur la structure intime du poumon de l'homme. Bruxelles, De Mortier frères, 1846.

OPSONIC INDEX FOR UVEAL PIGMENT IN TREATED PATIENTS

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This study was undertaken to determine the changes in the opsonic index in the blood serums of patients treated with uveal pigment. The rationale of this treatment has been fully described in various papers by A. C. Woods.¹ This author has shown that hypersensitivity to uveal pigment is a constant phenomenon in sympathetic ophthalmia and apparently paves the road for the outbreak of the disease, whatever other factors may initiate the process. Treatment with uveal pigment is designed to eradicate this hypersensitivity—to desensitize the patient. The clinical results of such treatments have been fully reported.² Likewise, there have been extensive studies on the antigenic properties of uveal pigment, demonstrating the organ specificity of this substance.³ There have, however, been no studies on the mode of absorption of uveal pigment in man. It was to determine this point that this study was undertaken.

Opsonins for granules of uveal pigment seemed important because extensive phagocytosis of pigment had been noted on biopsy in the intracutaneous uveal pigment and in the uveal tract of patients with sympathetic ophthalmia.⁴ Heat-stable opsonins for uveal pigment had never been demonstrated in human blood, though weak "normal" opsonins for beef pigment were demonstrated in normal human blood in 1908.⁵ Heat-stable "melanopsonins" and complement-fixing bodies

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Woods, A. C.: Allergy in Its Relation to Sympathetic Ophthalmia, New York State J. Med. **36**:67 (Jan. 15) 1936.

2. Woods, A. C., and Knapp, A.: The Therapeutic Use of Uveal Pigment in Sympathetic Ophthalmia, Arch. Ophth. **51**:560, 1922. Woods, A. C.: Sympathetic Ophthalmia: The Use of Uveal Pigment in Diagnosis and Treatment, Tr. Ophth. Soc. U. Kingdom **45**:208, 1925.

3. Woods, A. C.: A Contribution to the Anaphylactic Theory of Sympathetic Ophthalmia, Arch. Ophth. **46**:8, 1917; Ocular Anaphylaxis: III. The Rôle of Uveal Pigment, *ibid.* **46**:283, 1917; IV. The Antigenic Properties of Uveal Tissue as Shown by Complement Fixation, *ibid.* **46**:503, 1917; V. Experimental Iridocyclitis, *ibid.* **47**:161, 1918.

4. Friedenwald, J. S.: Notes on the Allergy Theory of Sympathetic Ophthalmia, Am. J. Ophth. **17**:1008, 1934.

5. Shattock, S. G., and Dudgeon, L. S.: Observations on Phagocytosis Carried Out by Means of Melanin, Proc. Roy. Soc., London, s.B **80**:165, 1908.

had been produced in 1909 by injecting granules of melanin into guinea-pigs.⁶ These melanoposonins were not studied further because in 1909 bacterial opsonins seemed of greater practical importance.⁷ Apparently, the only "inert" substances ever injected to produce opsonins⁸ were granules of melanin,⁷ globules of milk⁹ and erythrocytes.¹⁰ The present study adds uveal pigment of man to the list.

TECHNIC

In determining the opsonic index the classic Wright technic was used first and then a modified technic with heparinized blood. All the determinations were checked by both methods. These technics are as follows:

1. *Wright Technic.*—To a good suspension of pigment 0.3 cc. of the serum to be tested and 0.3 cc. of washed cells from a normal patient were added by means of a Wright pipet which was equipped with a rubber bulb. Thorough mixing was accomplished by repeated expression of the contents on a warm watch glass. The mixture was sealed in the pipet and incubated at 37 C. for fifteen minutes. Then the mixing was repeated, and a smear was made according to Wright's technic. Fifty neutrophils were examined for phagocytosis of granules of pigment. The average number of granules of pigment phagocytosed by one neutrophil was recorded as the phagocytic index.

2. *Technic with the Use of Heparinized Blood.*—To a good suspension of pigment 0.6 cc. of the heparinized blood to be tested was added, in a Wassermann tube. The tube was tapped gently and then incubated fifteen minutes at 37 C. The tube was again tapped gently, and a smear was made according to Wright's technic. The phagocytic index was determined as with Wright's technic.

MATERIAL FOR STUDY

The heparinized blood and the blood serum of treated patients were obtained from J. M., a white boy aged 14 who had sympathetic ophthalmia, and M. H., a white man aged 24 who also had sympathetic ophthalmia (patients 1 and 2).

The heparinized blood and the blood serum from patients not receiving pigment therapy were obtained from patients with a variety of ocular conditions for which various kinds of treatment had been given.

RESULTS

1. *Increase in the Opsonic Index.*—The blood serum of patient 2 showed the following increase in the opsonic index for pigment: Before

6. Ledingham, J. C. G.: Phagocytosis of So-Called Neutral Substances, *Ztschr. f. Immunitätsforsch. u. exper. Therap.* **3**:119, 1909.

7. Knapp, A.: On the Occurrence of Opsonins in the Aqueous Humor. *Arch. Ophth.* **38**:372, 1909.

8. Muir, R.: A System of Bacteriology, Medical Research Council, London. His Majesty's Stationery Office, 1931, vol. 6, p. 365.

9. Neufeld, F., and Händel: Beiträge zur Kenntnis der Wirkung verschiedener blutösender Gifte, insbesondere des taurocholsauren Natriums und der Seife, *Arb. a. d. k. Gsndtsamte.* **28**:572, 1908.

10. Hektoen, L.: On the Specificity of Opsonins in Normal Serum, *J. Infect. Dis.* **5**:249, 1908.

therapy was begun the index ranged between 4 and 8. After therapy was begun the indexes were 24 in the third week of therapy, 40 in the fourth week and 45 in the twelfth week. The complement fixation reaction for beef pigment changed from negative to strongly positive before the sixth week of therapy. Simultaneous tests made with a variety of control serums showed indexes between 4 and 8 and negative complement fixation reactions. Biopsies of intracutaneous pigment indicated that the hypersensitivity to uveal pigment was greatest after the first three weeks of therapy and that it was only moderate in the twelfth week of pigment therapy. A control test with intracutaneous india ink was, of course, negative.

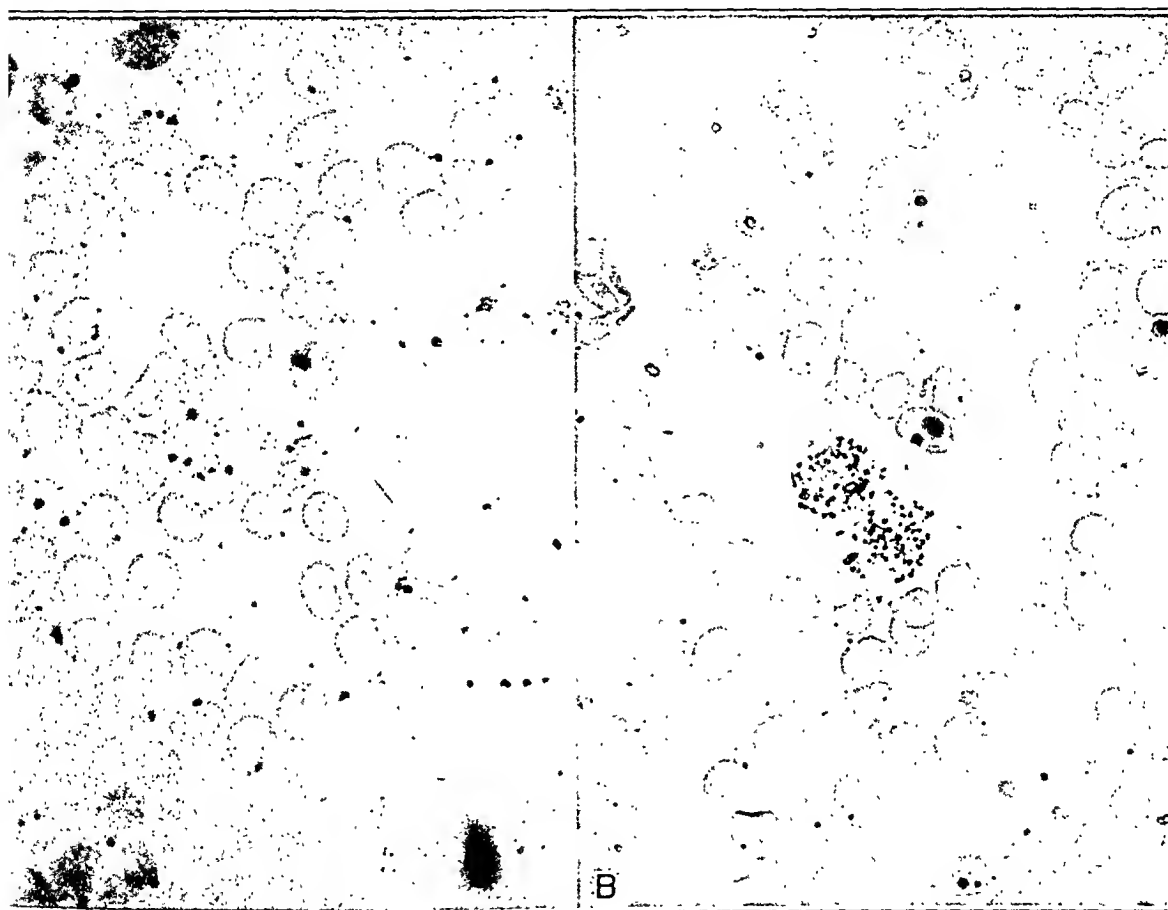


Fig. 1.—*A*, photomicrograph showing the influence of the serum of an untreated patient on the phagocytosis of pigment (human); *B*, photomicrograph showing the influence of the serum of a treated patient on the phagocytosis of pigment (human).

Patient 1 showed an index of about 45 for beef pigment during the last nine months of pigment therapy. He was followed for nine months after all therapy was stopped, and the index for beef pigment did not drop below 40. Intracutaneous tests showed that he was hypersensitive to pigment before therapy was begun but that he was not sensitive to pigment when therapy was stopped.

2. *Further Proof of the Organ Specificity of Pigment.*—In patients 1 and 2 beef pigment was used for the therapeutic injections. The opsonic index was determined not only for beef pigment but likewise for pork and human pigment. The therapeutic injections of beef pigment caused not only the increase in the index for beef pigment but an equal increase in the index for pork and human pigment.

Before pigment therapy was begun patient 2 showed indexes ranging between 4 and 8 for granules of beef, pork and human pigment when controlled tests were made simultaneously. In the twelfth week of therapy the indexes were 45 for beef pigment, 35 for pork pigment and 35 for human pigment. Another proof of the organ specificity of pigment was the marked phagocytosis noted in the biopsies of intra-cutaneous beef pigment and in biopsy of a prolapsed iris in this case. Extensive phagocytosis by epithelioid cells was prominent with the granules of beef pigment in the congested skin and with granules of human pigment in the prolapsed iris.

Nine months after pigment therapy was stopped patient 1 showed indexes of 40 for beef pigment and 30 for human pigment, in controlled tests carried out simultaneously. Since beef pigment was used in all the therapy, these findings were strong proof of the organ specificity of pigment. Control tests of patients not receiving pigment therapy uniformly gave indexes ranging between 4 and 8. The patients receiving milk therapy showed indexes above 6 for globules of milk, whereas all the other controls and patients 1 and 2 showed indexes below 2.

It was feared that the prozone phenomenon might invalidate the results obtained with the method in which heparinized blood was used. However, Wright's technic was used repeatedly with serum dilutions, and the prozone phenomenon could not be elicited.

To determine the heat-stable nature of the opsonins the following test was made with the serum of a patient treated with uveal pigment: The serum of this patient was heated at 56 C. for thirty minutes; normal complement was added, and the opsonic index for beef pigment was determined, by Wright's technic, to be 38. Control serums treated in the same fashion with heat and the addition of complement gave indexes below 4. The patient's serum showed an index of 42 when it was not heated and when complement was not added. The control serums showed indexes ranging between 4 and 8 when they were not heated and when complement was not added.

CONCLUSIONS

There is a unique increase in the opsonic index for uveal pigment in patients receiving pigment therapy.

The results obtained offer further proof of the organ specificity of pigment.

IS TRACHOMA A RICKETTSIAL DISEASE?

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TRANSLATED BY PHILLIPS THYGESON, M.D., NEW YORK

Researches on the etiology of trachoma have so often raised false hopes, of a solution of the problem that the ophthalmologist has become suspicious of new ideas. The investigator himself, if he is able to criticize his own researches, always finds some reason to question his results.

It is for this reason that I have placed a question mark after the title of this paper. Even if the recent experiments of Cuénod and Nataf¹ have confirmed the existence of the minute bodies which I described in 1933 in trachomatous tissues, particularly in the follicles, their nature and their rôle in the etiology of trachoma require still further study.

As a result of my researches,² which have recently been confirmed by Cuénod and Nataf, one sees looming on the obscure horizon of the etiology of trachoma a new category of germs, the rickettsias. Further investigations will tell whether the etiologic relation of these organisms to trachoma is a discovery or simply an invention.

In 1933 I reported before the Fourteenth Congress of Ophthalmology, held at Madrid,³ that I had observed in histologic sections and in smears of trachomatous material from the eyes of human beings bodies which by reason of their morphologic features and staining reactions I classified as rickettsias and to which I attributed a rôle in the etiology of trachoma. I arrived at this conclusion after many years of observation.

1. Cuénod, A.: Note préliminaire sur la présence d'éléments infra-microbiens dans les follicules trachomateux, *Arch. d'opht.* **52**:145 (March) 1935. Cuénod, A., and Nataf, R.: Deuxième note sur la présence d'éléments infra-microbiens dans les follicules trachomateux, *ibid.* **52**:573 (Aug.) 1935; Troisième note sur l'agent bactérien du trachome, *ibid.* **53**:218 (March) 1936.

2. Busacca, A.: (a) Ueber das Vorhandensein von Rickettsien-ähnlichen Körperchen in den trachomatösen Geweben und über das Vorkommen von spezifischen Veränderungen in Organen von mit Trachom-Virus geimpften Tieren, *Arch. f. Ophth.* **133**:41, 1934; (b) Un germe aux caractères de Rickettsies (*Rickettsia trachomæ*) dans les tissus trachomateux, *Arch. d'opht.* **52**:567, 1935.

3. Busacca, A.: Sulla presenza di germi simili a Rickettsie nei tessuti trachomatosi, *Klin. Monatsbl. f. Augenh.* **91**:277, 1933.

As early as 1925, in fact, when I was working at Bologna, I saw these bodies when staining trachomatous follicles according to the method of Jahnke for the demonstration of spirochetes in nerve tissues. But the little experience I had had at that time in the interpretation of preparations of this type and the criticisms of "official science" determined me to consider the results of these researches as negative and to place my observations "in quarantine."

I took up these studies again in 1931 in Brazil, where I had abundant pure material at my disposal. After applying the methods of silver impregnation in staining fragments of material from the eyes of patients with trachomatous pannus I observed in cases of successful impregnation the same bodies which I had seen in preparations of conjunctival material made in Italy.

The small size of these organisms and the frequent occurrence of dumb-bell-like forms did not permit them to be classified among the already known bacteria, but when I saw the figures illustrating the chapter on rickettsias by Rocha Lima in Kolle and Wasserman's "Handbuch der pathologischen Mikroorganismen" I was struck by the great morphologic resemblance between the rickettsias and the bodies which I had observed, and I continued my researches in this direction.

I tried to see whether by the methods appropriate for the demonstration of rickettsias one could not also stain these bodies that occurred in trachomatous tissues. After applying to smears and to histologic sections of tissues stains according to the method of Giemsa, of Castenada, of Lépine and of others I have been able to demonstrate them with great constancy. With a single exception they have been absent in the controls. This exception was a case in which I observed comparable bodies in the smears of material from a lymph gland of a patient with Hodgkin's disease.

The rickettsia-like bodies appear as small granules, round or slightly elongated, sometimes isolated, sometimes joined in dumb-bell form and sometimes in masses.

The characteristic dumb-bell form (all the stains do not permit one to see the small segment which unites the two granules) has a length varying from 0.5 to 0.8 micron and a width of approximately 0.2 micron.

I work with impression smears. After excising with the scissors, in the region of the upper fornix, a fragment of conjunctiva rich in follicles, I hold the piece with a forceps and proceed to make light impressions on the slide; when the fragment leaves no more material I crush it a little between the arms of the forceps and continue the procedure.

The smears so obtained show some zones which are too thick and not utilizable, but, on the other hand, this technic offers the great

advantage of avoiding the rupture of a large number of nuclei and permits the study of cells of the deeper layers, which are present rarely in scrapings.

In the smears the bodies are stained violet-red by the Giemsa stain at p_H 7.2, blue by the method of Lépine and of Castenada, and blue by the victoria blue stain according to the method of Muehlpfordt, Kiel and van der Gerge. Last year I obtained some beautiful results by applying Herzberg's victoria blue method for the demonstration of viruses,⁴ and I am of the opinion that for rapidity, simplicity and sharpness of the images obtained this method can be employed as a routine for diagnosis. With it one can easily see around the rickettsias a



Fig. 1.—Epithelial cells with numerous rickettsias in the protoplasm; smear stained with victoria blue; \times 1,410.

minute halo, not always demonstrable by the other methods, and can note in a single cell certain differences in size and staining reactions among the rickettsias. Often it is possible to see masses which may represent multiplication forms.

The bodies are seen in epithelial cells, in cells of the follicle or even in a free state between the cells. In the cells they are sometimes scattered and sometimes grouped; occasionally they may fill the cell completely.

The free forms are not always identifiable and cannot be considered of great significance, but when they appear as small agglomerations

4. Busacca, A.: Méthode simple et rapide au bleu victoria pour la démonstration des rickettsies du trachome, *Folia clin. et biol.* 7:253, 1935.

between the lymphocytes they may be recognized easily; between the epithelial cells they often form short filaments.

In histologic sections the demonstration of the rickettsias is more difficult. The method of Jahnke and the victoria blue stain, however, give beautiful preparations. Since the publication of my work I have also succeeded, in sections stained by the Giemsa stain, in staining the rickettsias red by adding some colophene to the alcohols used for differentiation. Some intracellular and some extracellular forms occur in the epithelium and in the tissues of the follicle. In the most superficial layers of the corneal epithelium one often sees cavities filled by bodies which, according to the stain employed, appear as very fine



Fig. 2.—Corneal epithelium in which the basal cells contain rickettsias; histologic section prepared according to the method of Jahnke; $\times 1,410$.

granules or as a network of light threads along which darker granules are disposed. In certain cases the method of Jahnke shows ring forms.

The staining characteristics of these bodies may be summarized as follows:

In smears they stain violet-red with the Giemsa stain at pH 7.2, light blue with the method of Castenada, blue with the method of Lépine, blue with victoria blue according to the method of Muehlpfordt, Keil and van der Gerge, and shades varying from blue to violet-blue with Herzberg's victoria blue stain.

In sections they stain red with the Giemsa stain, red or violet-red with Mann's method, deep blue with victoria blue, and light red after

prolonged staining with eosin and differentiation. They are very pale when stained with iron hematoxylin.

The morphologic features and the staining properties are thus seen to correspond to those of the rickettsias.

In a report published in 1934 Thygeson⁵ attributed the characteristics of rickettsias to the elementary corpuscles of the Prowazek bodies; furthermore, like the majority of authors, he stated that he had never encountered them in the cells of the follicles but had seen them only in the epithelium. In the course of my researches I have always had the impression that the bodies which I have described differed from the elementary bodies. The examination of smears sent me by Thygeson has confirmed my opinion on this point, and Thygeson has concurred in it after examining the smears which I sent him. He

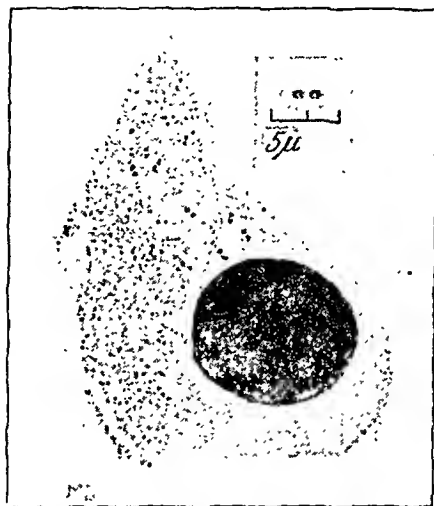


Fig. 3.—Epithelial cell with rickettsias scattered in the protoplasm; smear stained with the Giemsa stain; $\times 1,410$.

observed the bodies which I described also in cases of trachoma in North America, but he has stated that he is not sure of their rickettsial nature and thinks that sufficient reasons have not been advanced to enable one to conclude that they are living organisms.⁶

Very interesting, according to my point of view, are the observations published by Cuénod and Nataf,¹ in which these authors referred to the presence of rickettsia-like bodies in smears from the contents of trachomatous follicles and in histologic sections from these follicles. The description which figured in the transactions of the Fourteenth International Congress of Ophthalmology, held at Madrid, and the iconographic documentation which I have given in publishing a com-

5. Thygeson, P.: The Nature of the Elementary and Initial Bodies of Trachoma, *Arch. Ophth.* **12**:307 (Sept.) 1934.

6. Thygeson, P.: Personal communication to the author.

plete account of my researches leave no doubt of the identity of that which I have observed and of that which Cuénod and Nataf have described. These authors have, in addition, clearly recognized in their reports the priority of my observations.

The use of appropriate methods (such as the cyanochine method) for obtaining negative images has made it possible for Cuénod and Nataf to evaluate the large numbers of bodies scattered in the smears; their interpretation remains doubtful when other methods are used. But these authors have succeeded in extending their observations beyond the morphologic field and recently reported some experimental researches⁷ the results of which, if they are confirmed, will show clearly

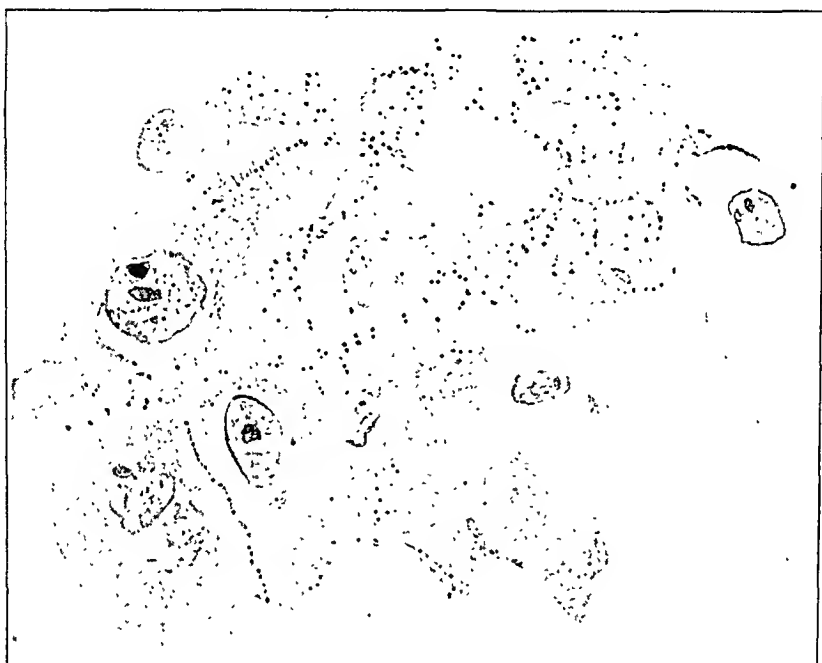


Fig. 4.—Epithelial cells of a cornea affected with pannus, showing intracellular and intercellular rickettsias; histologic section stained with the Giemsa stain; $\times 1,410$.

the nature and rôle of these bodies. After inoculating some trachomatous material into the louse they observed in the epithelial cells of the intestine of these insects rickettsia-like bodies absolutely identical with those observed in the cells of the trachomatous follicles which they studied, and by the inoculation of material from infected lice they have been able to reproduce in the monkey a form of conjunctivitis having the characteristics of experimental trachoma.

7. Cuénod, A., and Nataf, R.: Nouvelles recherches sur le trachome: IV. Recherches expérimentales, *Arch. d'opht.* **53**:335, 1936.

The demonstration of these bodies is not easy, and Cuénod and Nataf have expressed the opinion that perhaps "because of inability easily to demonstrate them their existence will not be accepted." I believe that staining fresh smears with victoria blue according to the method of Herzberg is the simplest means of demonstrating them, but this stain is no more specific than the other stains, and I fear very much that the difficulties of demonstration may lead to false interpretations, as has occurred in connection with the Prowazek bodies.

EXPERIMENTAL RESEARCHES

In an attempt to give experimental confirmation to my observations I have tried the inoculation of trachomatous material into various



Fig. 5.—Large mononuclear cell of a trachoma follicle; few rickettsias are seen in the protoplasm; smear stained with the Giemsa stain; $\times 1,410$.

organs (testicle, brain, spleen, lymph gland, vitreous body and anterior chamber of the eye) of the guinea-pig, the rabbit and the white mouse.⁸

In the testicle of the guinea-pig I obtained some reactions which I considered specific: In the cells of the vaginal sheath of the testicle I observed some rickettsia-like bodies. By intravitreal inoculation I also obtained some reactions which I at first considered specific but on the interpretation of which I was later led to modify my opinion. With the exception of the one case already cited I never have succeeded in demonstrating the rickettsia-like bodies either in the eyes or in any of the other inoculated organs.

While pursuing my researches with intravitreal inoculation I had observed in the chicken the formation of intra-ocular lymphatic nodules

8. Footnotes 2a and 3.

when von Szily⁹ wrote (in January 1935) that he had obtained, as a result of intravitreal inoculation of an emulsion of trachomatous conjunctiva, the formation of follicles, which he believed to be trachomatous, in the interior of rabbits' eyes atrophic from severe uveitis. The publication of this work preceded that of my researches¹⁰ by several weeks (the results of my work were published in March 1935). Other reports on the same subject have since been published by von Szily in the *Klinische Monatsblätter für Augenheilkunde* in 1935 and 1936.

Because of the occurrence of a certain number of cases of bacterial contamination in the direct inoculation of trachomatous material I conceived the idea of avoiding this danger by treating the material used for inoculation with glycerin; this would also test the resistance of the agent of trachoma to glycerin.

In May 1935 I communicated to the Société de biologie¹¹ that I had observed, after the intravitreal inoculation of glycerinated tra-

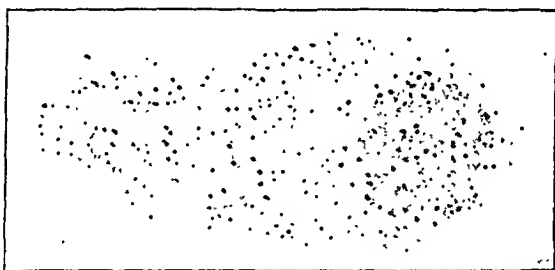


Fig. 6.—Large mononuclear cell of a trachomatous follicle with rickettsias in the protoplasm; smear stained with victoria blue; $\times 1,410$.

chomatous material into the eyes of the rabbit and the chicken, the appearance of intra-ocular nodules; nevertheless the questions which arose as to the interpretation of my previous experiments have led me to speak of the presence in trachomatous tissue of a virus resistant to glycerin and not of the resistance of the trachoma virus to this alcohol.

Suspecting, from retrospective examination of my results, that the factor "quantity of injected material" could play a rôle in the production of these nodules, I proceeded with tests on controls. I saw intra-

9. von Szily, A.: Uebertragungsversuche mit Trachommateriel; Ein weiterer Beitrag zur Kenntnis follikelbildender Erreger, *Klin. Monatsbl. f. Augenh.* **94**: 1 (Jan.) 1935.

10. Busacca, A.: Nouveaux résultats en pathologie expérimentale du trachome, *Compt. rend. Soc. de biol.* **120**:167, 1935.

11. Busacca, A.: Sur la présence dans les tissus trachomateux d'un virus résistant à la glycérine et transmissible en série aux animaux, *Folia clin. et biol.* **7**:63, 1935.

ocular nodules appear after the intravitreal inoculation of a very heavy glycerinated emulsion of normal intra-ocular membranes. Basing my opinion on the fact that one obtains intra-ocular nodules by the intravitreal inoculation of different materials (von Szily noted such nodules after the inoculation of material from eyes enucleated for sympathetic ophthalmia and after the inoculation of trachomatous conjunctivae, and I noted similar nodules after the inoculation of fragments of cornea affected with trachomatous pannus, of trachomatous conjunctiva conserved from eight to fifteen days in glycerin or of normal glycerinated material), I have come to the conclusion that the nodular reaction of intra-ocular tissues ought to be considered as a reaction peculiar to this tissue and not as a reaction to a category of germs.¹² Probably one should attribute to these nodules the same significance as to those (lymphatic follicles) observed in certain experimental conditions by other authors (Greggio¹³ observed lymphatic follicles in the interstitial tissue of the kidney of the rabbit after staphylococcic infection; Lang¹⁴ noted such follicles in the mesentery of rabbits treated with phenylhydrazine or sapotoxin, and other investigators have made similar observations). They are, moreover, of the same type as those described by Zimmermann¹⁵ and others in atrophic eyes and by Woods and Chesney¹⁶ in eyes inoculated with material from the eyes of horses with moon blindness.

In regard to the demonstration of rickettsia-like bodies in the experimental material, my researches have not, up to now, given very satisfactory results. As I have stated, such bodies were demonstrated with relative ease in the cells of the vaginal sheath of the testicles of inoculated guinea-pigs. On the other hand, I never have observed them in the endothelium of the cornea after the inoculation of trachomatous material into the anterior chamber, in smears from various intra-ocular tissues after intravitreal inoculation or even in eyes in which nodules had developed. Finally, in one single case of a fowl in which an imposing nodular conjunctivitis developed after the inoculation of

12. Busacca, A.: Sur la valeur des nodules qu'on obtient après inoculation intravitréenne de matériel trachomateux, *Folia clin. et biol.* **7**:180, 1935.

13. Greggio, L., cited by Maximow: Bindegewebe und blutbildende Gewebe, in von Möllendorf, W.: *Handbuch der mikroskopischen Anatomie*, Berlin, Julius Springer, 1928, vol. 2, p. 373.

14. Lang, cited by Maximow: Bindegewebe und blutbildende Gewebe, in von Möllendorf, W.: *Handbuch der mikroskopischen Anatomie*, Berlin, Julius Springer, 1928, vol. 2, p. 373.

15. Zimmermann, S.: Ueber Rundzellenherde in der Uvea atrophischer Augen, *Ztschr. f. Augenh.* **57**:279, 1925.

16. Woods, A. C., and Chesney, A. M.: The Transmission of Periodic Ophthalmia of Horses by Means of a Filterable Agent, *J. Exper. Med.* **52**:637, 1930.

glycerinated trachomatous material I was able to demonstrate some rickettsia-like bodies in smears made from fragments of the nictitating membrane.

CONCLUSIONS

Such are the facts observed in the course of these last years by me and by other authors in the domains of the etiology and experimental pathology of trachoma. From these facts some new ideas have been introduced into the study of this chapter of ocular pathology which is still surrounded by mystery. The researches are too recent and too few as yet to permit the drawing of definite conclusions. It is better to wait until further investigations clarify the questions relative to this interesting problem.

METASTATIC PNEUMOCOCCIC UVEOSCLERITIS FOLLOWING PNEUMONIA

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Metastatic infections of the inner ocular structures are not rare. The septic embolus is usually carried into the eye either through the retinal or through the uveal system of blood vessels. Experiments of Selenkowsky and Woizechowsky¹ demonstrated that the embolus usually lodges in the posterior segment of the eye, i. e., in the retina (if carried by the central retinal artery) or in the choroid (if carried by one of the short posterior ciliary arteries).

Infections of the anterior segment of the eye, i. e., of the iris and ciliary body, are usually supposed to be transmitted by the long posterior ciliary arteries. These arteries enter the interior of the eye near the posterior pole and run, one on the nasal side, the other, on the temporal side, of the eyeball, between the choroid and the sclera, as far as the ciliary muscle. At the root of the iris each divides into two branches which run in a direction concentric to the limbus and form the major arterial circle of the iris. The common type of infection transmitted through this path is an inflammation of the iris and ciliary body designated by Friedenwald² as subacute iridocyclitis, in contradistinction to acute purulent iritis or cyclitis, which is characterized by formation of a localized abscess. The latter type of inflammation is usually accompanied by a hypopyon or even by a corneal ring abscess which may result in perforation of the cornea.

The anatomy of the uveal system of blood vessels suggests a possibility of one or more types of metastatic infection of the anterior ocular segment, namely, transmission of the embolus through one of the anterior ciliary arteries. These arteries, usually seven in number, are branches of the muscular arteries and pierce the sclera between the attachments of the muscular tendons and the limbus. They supply the ciliary body and communicate with the major arterial circle of the iris.

In the case which is reported in this paper the patient showed a violent inflammation of the sclera and of the uvea, simultaneously with formation of an abscess between the limbus and the tendon of the

1. Selenkowsky, J., and Woizechowsky, N.: *Experimental Work on Metastatic Panophthalmitis*, Arch. f. Augenh. **47**:299, 1903.

2. Friedenwald, Jonas S.: *The Pathology of the Eye*, New York, The Macmillan Company, 1929, p. 44.

superior rectus muscle. Such a location of an abscess could be explained by lodging of a septic embolus in one of the scleral canals formed by the anterior ciliary arteries.

Intra-ocular endogenous infections are often caused by pneumococcus. In the monograph of Seguni³ 24 cases of ocular metastatic infection due to pneumococcus are reported and contrasted with 29 cases of ocular metastatic infection of other bacterial origin. The same monograph, however, contains in addition a description of 250 cases of endophthalmitis of undetermined bacteriologic origin.

The first description of pneumococcic metastatic endophthalmitis was published by Axenfeld⁴ in 1894. In one of his cases the condition was traced to hematogenous infection, and in the other, in which the disease followed pneumococcic meningitis, the patient showed bacterial infiltration along the arachnoid spaces into the optic nerve and into the eyeball. Complete clinical and pathologic descriptions of pneumococcic endophthalmitis were published by Bussola,⁵ Lawson⁶ and Heinomen.⁷ In the case reported by Bussola the condition was bilateral. The left eye became affected on the seventh day of pneumonia. The ocular symptoms started with iritis in the left eye, which was rapidly followed by a ring abscess of the cornea and resulted in perforation of the cornea six days later. The right eye presented symptoms of endophthalmitis four days later than the left eye, and a ring abscess developed twenty-four hours before death from meningeal complication. In the case reported by Lawson the condition followed pneumococcic otitis media. It was also characterized by a ring abscess and perforation of the cornea. In the case reported by Heinomen the condition followed an attack of common cold, and the ocular embolus appeared with the beginning of menstruation. The eye had to be eviscerated.

In none of the reports just cited did the location of the abscess correspond with that in my case.

Descriptions of metastatic scleritis do not tally any better. Nodular scleritis is usually ascribed to focal infection, without determination of the bacteriologic features, to tuberculosis or to syphilis. This disease

3. Seguni, A.: Coroidite metastatica, *Ann. di ottal. e clin. ocul.* **7**:301 (April) 1923.

4. Axenfeld, T.: Ein weiterer Beitrag zur Lehre von der eitrigen metastatischen Ophthalmie, *Arch. f. Ophth.* **40**:4, 1894.

5. Bussola, E.: Bilateral Pneumococcic Infection of the Eye in Pneumonia, *Boll. d'ocul.* **10**:987 (Sept.) 1931.

6. Lawson, L. J.: Metastatic Panophthalmitis Following Pneumococcic Otitis Media, *J. A. M. A.* **101**:599 (Aug. 19) 1933.

7. Heinomen, Oskar: Case of So-Called Spontaneous Panophthalmia, *Finska läk.-sällsk. handl.* **74**:360 (April) 1932.

has a more protracted course and less fulminant symptoms. So-called malignant scleritis of von Hippel, which is accompanied by intra-ocular complications, is of tuberculous origin. Robert Hesse⁸ examined one patient microscopically and found that the intra-ocular involvement was localized mainly in the choroid and only in a lesser degree in the ciliary body. In my case the main intra-ocular involvement was in the ciliary body. Besides that, the condition in my case was definitely of pneumococcic origin, while in the case of Hesse it proved to be tuberculous.

According to Selenowsky and Woizechowsky,¹ the actual occurrence of intra-ocular metastatic infection depends on the degree of bacteremia and on the presence of trauma to the eyeball. This contention is supported by the deduction of Seguni³ that toxic lesion of the arteries is necessary for the development of an ocular metastasis. Heinonen⁷ ascribed the ocular metastasis of his patient to premenstrual constriction of capillaries as described by Hagen.⁹ None of these etiologic factors was present in my patient. The ocular symptoms started in the intermenstrual period. There was no history of trauma to the affected eye. The healthy eye was under constant observation, and no vascular changes could be seen in the retina, choroid or ciliary vessel. It can be assumed that vascular or retinal changes would be noticeable also in the healthy eye if such changes were contributory to the development of the metastasis in the affected eye.

It was probably an anatomic factor which determined the localization of the embolus, namely, narrowing of the artery and rigidity of the tissues surrounding the anterior ciliary artery as it passes through the sclera.

REPORT OF A CASE

History.—M. W., a white woman, a widow, aged 40 years, was admitted to the St. John's Long Island City Hospital, Long Island City, N. Y., on May 25, 1935, for lobar pneumonia of the right lung. The previous history and the family history were irrelevant. A blood culture taken on June 6 showed pneumococci which did not react with specific antisera types I, II, III, V, VII and VIII. The disease had the usual febrile course and ended on June 10 with resolution. On June 12 the left eye appeared red and was very painful, and the patient's sleep was disturbed. On June 14 I was called for consultation regarding the ocular condition.

Examination of the Eyes.—This was carried out on June 14.

Right Eye: Vision was 20/20. External examination gave negative results. The cornea was clear. The iris was blue, and the pupil was narrow and regular and showed a prompt light reflex. The media were clear. The fundus was without pathologic changes. The tension was 17 (Schiötz).

8. Hesse, R.: Zur pathologischen Anatomie der Skleritis, Ztschr. f. Augenh. 83:324 (July) 1934.

9. Hagen, W.: Tuberkulose und Kapillarsystem, München. med. Wchnschr. 70:532 (April) 1923.

Left Eye: Vision was perception of light; projection was good. Both lids were swollen, and the conjunctiva of the bulbus was swollen and diffusely red. The swelling extended into the lower fornix. The circumcorneal vessels were injected and purplish. The cornea was steamy. The anterior chamber was deep. The pupil was about 2 mm. in diameter, irregular and filled with light grayish exudate. Posterior synechiae were present around the whole pupillary margin. Iris bombé was present; the iris was greenish. A corneal microscope was not available, but on examination with the corneal loupe Descemet's membrane did not show any deposits. The fundus reflex could not be obtained. The tension was 35 (Schiötz). Practically equal luminosity of both pupils was obtained by transillumination from all directions.

There was an extremely hypersensitive area in the sclera located between 12 and 3 o'clock in the ciliary zone. The patient localized the pain in this segment even without touching the eyeball. However, no increased infiltration or congestion could be observed in the painful area, compared with other parts of the sclera.

The lacrimal sac and puncta were normal.

There was no evidence or history of trauma.

Binocular excursions of the muscles in six cardinal directions were not impaired and caused no pain.

General Physical Examination.—The results of the physical examination had been recorded on the previous day by Dr. E. W. McLave. Examination had revealed a few moist râles in the region of the right hilus. The ears and nose showed no pathologic changes. The pulse rate was 90 in the afternoon. The temperature was 99.8 F. The action of the heart was regular, and the sounds were clear. The blood pressure was 125 systolic and 80 diastolic.

Treatment.—This consisted of instillations of a 4 per cent solution of atropine sulfate and hot applications over the left eye, sodium salicylate in solution, in doses of 1 Gm., three times a day, and intramuscular injections of sterile milk protein.

Operations and Course.—Atropine failed to disrupt the synechiae and to dilate the pupil. The iris bombé was not reduced. Swelling of the conjunctiva gradually increased, and on June 16 the swollen conjunctival and episcleral tissues started to protrude between the eyelids. The cornea remained hazy and the tension continued to be around from 35 to 40 (Schiötz). The pupillary exudate did not increase. Pain was very severe. Perception of light was not lost, and luminosity on transillumination was not noticeably decreased.

On June 18 paracentesis of the anterior chamber was performed with the patient under local anesthesia. Retrobulbar injection of a 2 per cent solution of procaine hydrochloride was carried out. The chamber was entered with a cataract knife through the swollen conjunctiva and through the limbus from the temporal side, a little above the equator of the cornea. The bowed iris was transfixated in four places, and the point of the knife was allowed to emerge through the limbus under the swollen conjunctiva of the nasal side. During the withdrawal of the knife a piece of iris prolapsed between the lips of the sclerocorneal incision on the temporal side. No attempt was made to replace the prolapsed portion because it was well covered by the swollen conjunctiva. The operation was followed by immediate relief of pain.

On June 19 the cornea was clear; the anterior chamber had reformed and contained clear aqueous, and a small amount of viscous exudate was present in the pupil. The iris was flat. Edema of the conjunctiva increased enormously. The

swollen lower fornix protruded and covered the lower eyelid down to the palpebro-malar fold. Measurement of tension with the tonometer was no more possible because the corneal area which remained uncovered by the periconjunctival swelling was too small to admit the contact disk of the instrument. Palpation through the upper eyelid indicated about normal tension. The patient felt comfortable and had no pain.

On June 20 the upper conjunctival cul-de-sac started to protrude. The prolapsed portion had a mushroom-like formation and covered the upper tarsus from the outside. After retraction of the prolapsed parts the visible segment of the cornea appeared clear; the anterior chamber was deep, with the same amount of central exudate as on the day before.

The prolapsed parts were covered with a bandage to which petrolatum had been applied and were irrigated several times a day with hot saline solution.

The condition continued unchanged until June 27, when the upper part of the prolapsed portion began to recede. The patient was fairly comfortable all the time and did not suffer pain.

On June 30 the cornea was again visible. The exudate in the pupil was flat and began to organize. The tension was 15 (Schiötz). The lower fornix was still prolapsed and covered the lower eyelid. Perception of light was present, and projection was good.

Between July 1 and July 8 the mushroom-like protrusion of the upper fornix disappeared, and the upper eyelid again covered the eye. The lower fornix remained prolapsed.

On July 9 a nodular infiltration was observed in the sclera above the upper pole of the cornea. The depth of the anterior chamber began to diminish, and the aqueous became cloudy.

The infiltration was rapidly enlarging and on July 12 was about 1 cm. in diameter, soft, fluctuating and yellow. It was incised with the patient under local anesthesia, and about 0.5 cc. of yellow pus was evacuated. The bottom of the abscess was probed. It appeared soft and raggy and did not have the usual consistency of sclera. No fistula leading into the eyeball was discovered. Culture of the pus revealed pneumococci identical with those obtained from the previous blood culture.

After the evacuation of the pus the eyeball was very soft, and the remaining edema of the conjunctiva rapidly diminished.

On July 15 the patient was discharged from the hospital. On the day of discharge perception of light was still preserved; projection into the nasal field was doubtful. The anterior chamber was shallow, and the eye was very soft.

Through the whole course of this ocular complication the temperature alternated daily between 98.5 and 100.5 F.; it returned to normal after evacuation of the pus. No other purulent foci were observed in other organs. The right eye did not show any symptoms of inflammation or irritation.

After discharge from the hospital the patient was observed in the office. Within about four weeks the affected eyeball had shrunk, and perception of light was lost. The eye was finally enucleated on March 16, 1936, with the patient under general anesthesia. The site of the abscess was marked by insertion of a black silk suture into the sclera, and the eyeball was sectioned by Dr. Joseph Levine in the Manhattan Eye, Ear and Throat Hospital. The microscopic examination was made by Dr. Levine. The report, which was made on April 27, is as follows:

Microscopic Examination.—The specimen was received in March 1936.

The globe was shrunken and deformed. The cornea was irregularly wrinkled, and the layers of epithelial cells were irregular in thickness. Some of the basal cells were vesicular.

There were many blood vessels throughout the cornea. The lenticular space was filled with a homogeneous granular fluid; the cells of the anterior capsule had proliferated.

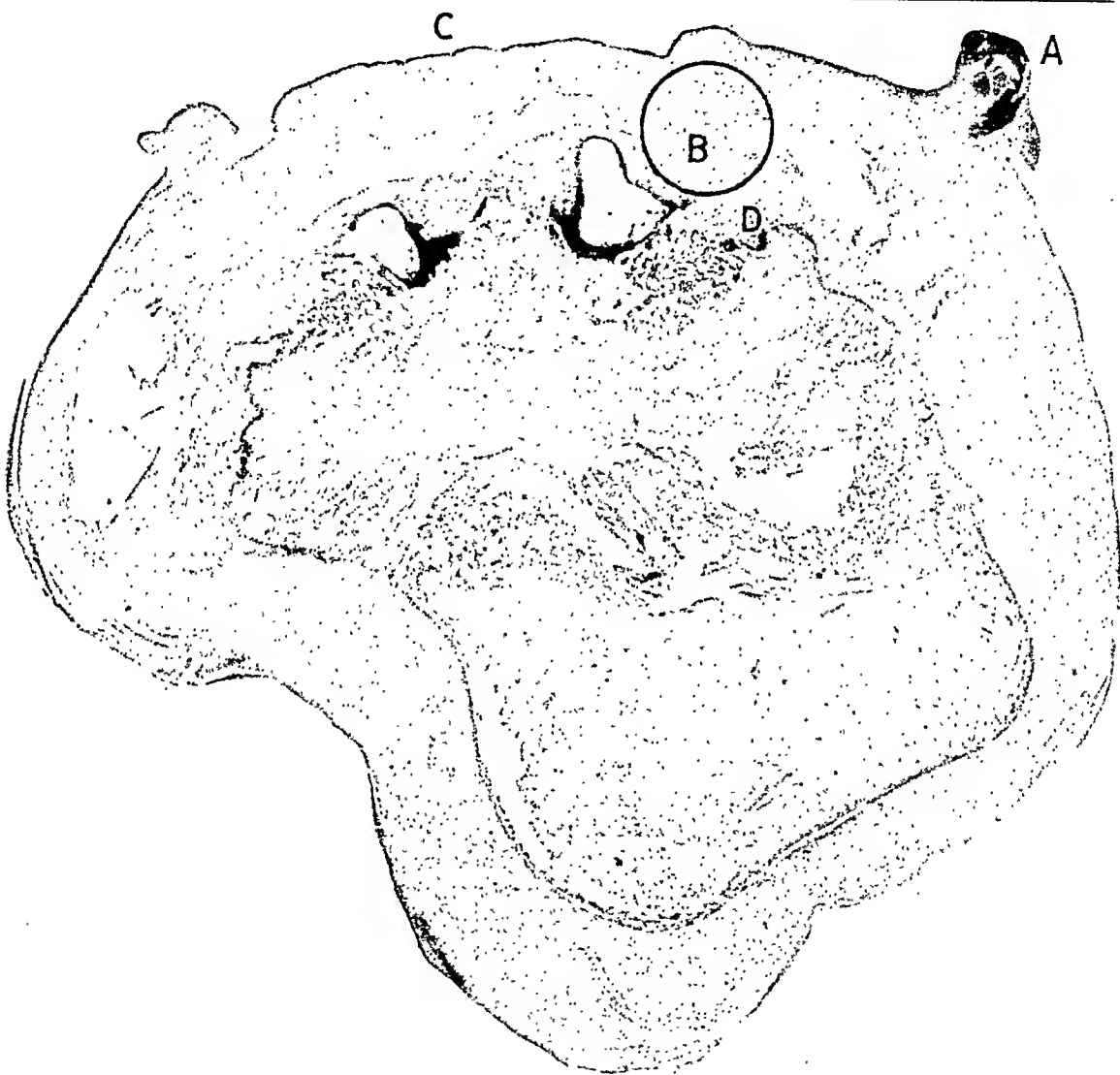


Fig. 1.—Section showing metastatic pneumococcal uveoscleritis resulting in phthisis of the eye. *A* shows the black silk suture inserted above the healed scleral abscess. *B* is the area shown enlarged in figure 2. *C* indicates the cornea, and *D*, the ciliary body.

The iris was enormously thickened by an infiltration with inflammatory cells, and the pigment epithelium had proliferated.

Descemet's membrane was markedly thickened and was folded on itself in many places. The anterior chamber contained an albuminous exudate. The ciliary body and processes were distorted and also contained many inflammatory elements. In the region of the ora serrata the remains of the completely detached retina were folded over, and there were many pus cells.

The choroid was wrinkled, as in cases of long-standing detachment of the retina. The space between the choroid and the detached retina was occupied by a

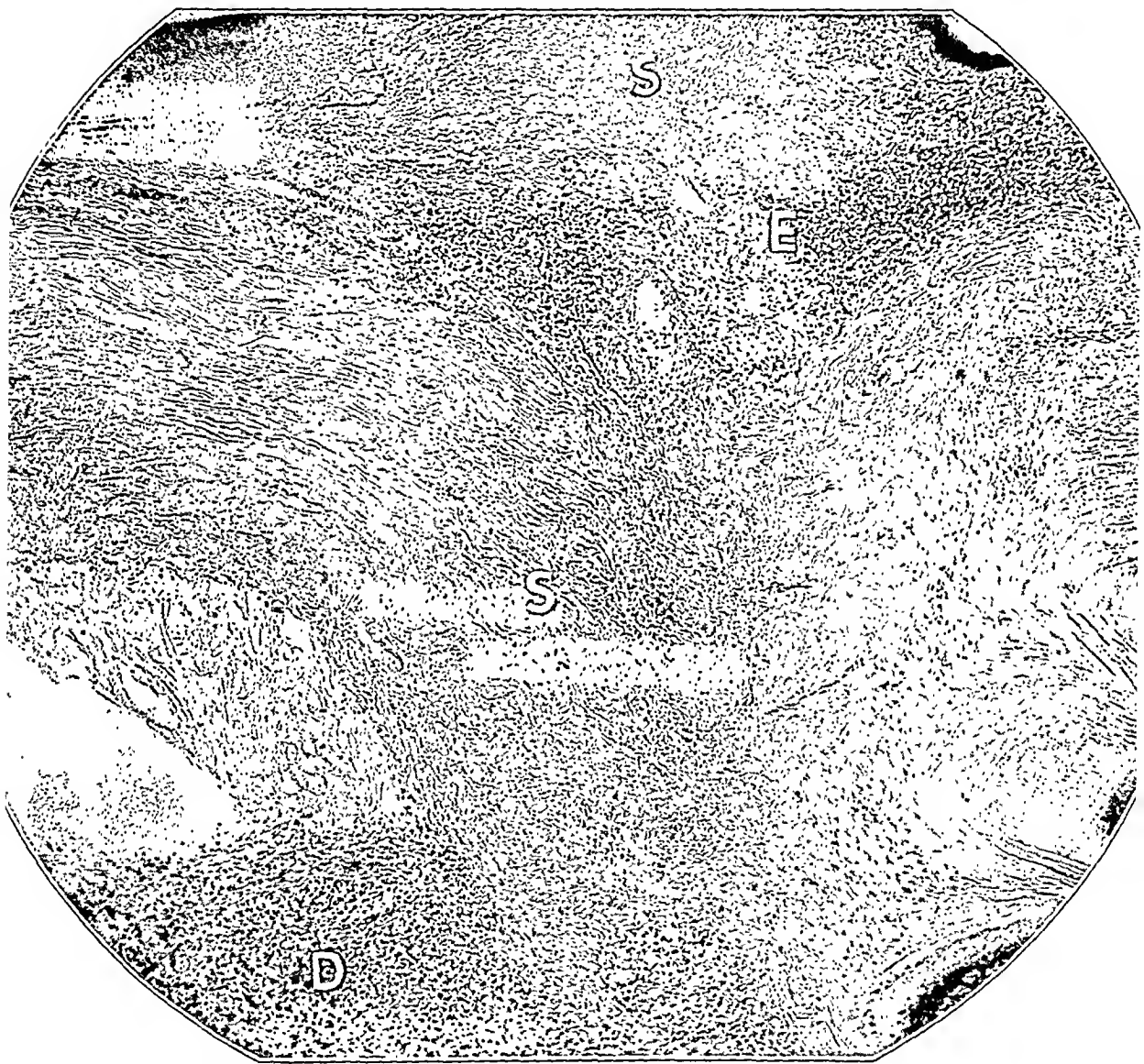


Fig. 2.—Photomicrograph of section showing the scar in the sclera connecting the infiltrated ciliary body, *D*, with the episcleral tissue, *E*. *S* indicates the sclera, which is wrinkled in the upper part of the section.

homogeneous albuminous exudate, somewhat similar to that seen in the anterior chamber. There was no doubt that infection was localized in the anterior segment of the eye, that is, the ciliary body and iris.

Pathologic Diagnosis.—The diagnosis made on the basis of the pathologic examination was phthisis bulbi following endophthalmitis.

COMMENT

The condition in this case can be classified neither as simple metastatic endophthalmitis nor as simple metastatic scleritis, for the following reasons :

1. From the onset of the ocular complication the tenderness was localized in the ciliary region above the upper pole of the cornea, without signs of localized scleral infiltration.

2. The symptoms of intra-ocular inflammation were comparatively mild, as manifested by good projection and perception of light and by normal transillumination until phthisis set in.

3. Primary involvement of the uveal tract was manifested by the rapid formation of pupillary occlusion at the very onset of the disease.

4. The enormous swelling of the conjunctiva and of the episcleral tissue points to simultaneous scleral involvement.

5. There was only a small amount of pus evacuated from the abscess, which did not communicate with the vitreous chamber.

6. Section of the eye showed organization in the anterior half of the eyeball, the posterior half having remained relatively intact (fig. 1).

7. Infiltration of the sclera is still visible on the section. It formed a path leading from the ciliary body through the sclera into the episcleral tissue (fig. 2).

It can be assumed that a septic embolus lodged in one of the anterior ciliary vessels as it transfixes the sclera on its course from the muscular artery to the ciliary body.

Therefore this case should be classified as one of metastatic uveo-scleritis following pneumonia.

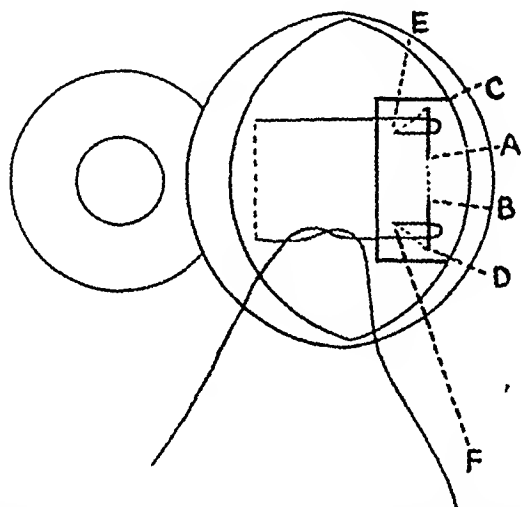
Clinical Notes

A SINGLE SUTURE ADVANCEMENT OPERATION

GUY A. HUNT, M.D., BUTLER, PA.

This operation is submitted as my modification of Prince's advancement operation, particularly regarding the insertion of the suture in the tendon of the muscle.

A vertical incision is made in the conjunctiva about 6 mm. from the limbus. After the conjunctiva is undermined up to the limbus and over the tendon of the muscle, Tenon's capsule is buttonholed at one side of the tendon, a strabismus hook is inserted under the tendon, and Tenon's capsule is incised on each side but not stripped from over the tendon. A Prince's advancement forceps is now applied near the insertion of the tendon, with the handle of the forceps toward the cornea, and the tendon is cut close at its insertion.



Technic of the single suture advancement operation.

I use a double-armed suture of 000 ten day chromacized catgut and substitute for one of the atraumatic needles (which come with the suture) a Jameson needle to be used in picking up the scleral fibers.

The needles are first entered at *A* and *B* from the scleral surface of the tendon so as to include the middle quarter of its width between these two points. One needle is now passed inward through the tendon 1 mm. or less from the upper border at *C* and entered again from the scleral surface at *E*, 2 or 3 mm. from the loop *AC* and equidistant from the points *A* and *C*. The same needle is next passed under and then over the loop *AC*, which should then be drawn close to the tendon by traction on the suture near *E*. The other needle is used in a like manner to place the suture in the lower portion of the tendon. After the loops are drawn up closely the ends of the suture are held by an assistant, and the tendon is resected between the forceps and the suture.

In the next step the conjunctiva near the limbus is retracted, and one of the needles is passed under the superficial fibers of the sclera about 1 mm. from the limbus in a vertical direction and with divided bights for a distance of 6 or 8 mm. The suture is then carefully drawn up so as to approximate the end of the tendon with the sclera near the limbus and tied. The conjunctiva is sutured with silk or plain catgut.

The three advantages claimed for this operation are: (1) assurance that the suture will not pull out of the tendon, since the loops tighten with an increase in traction; (2) a strong scleral attachment; (3) proper alinement of the muscle.

The argument that single suture advancement operations cause pointed or contracted attachments does not hold true for the operation just described, since the sides of the tendon do not curl under and the end is held in firm contact with the sclera to the extent of the width of the tendon. The correctness of this may be demonstrated by inserting the suture in a piece of tape $\frac{3}{8}$ inch (9.6 mm.) wide. Also, with the same tape it may be shown that if the points of insertion are near the borders in following Prince's method the sides of the tape will curl under so as to contract the width of the end; but by making the loop to include the middle fourth such curling does not occur. In the operation described I have therefore included only the middle fourth of the tendon in the central loop.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

GENERAL ANESTHESIA IN OPERATIONS ON THE EYE, EAR, NOSE AND THROAT

JOHN S. LUNDY, M.D.

ROCHESTER, MINN.

The administration of general anesthetics by inhalation for operations on the eye, ear, nose and throat has been rather well standardized in the past. The commonest method has been the induction of anesthesia by nitrous oxide or ethyl chloride and its maintenance by ether administered by the drop method followed by insufflation of ether vapor from the Junker bottle or from some mechanical insufflator if the operation is that of tonsillectomy or some other procedure on the throat. When a gas machine was available, a gas, such as ethylene, with or without ether, has been used extensively, but since this article is purposely limited to those recent advances which have allowed the surgeon an increasing choice in the methods of operations the older methods of inducing anesthesia are referred to only briefly.

The use of a Magill large bore soft rubber intratracheal tube has made the administration of anesthetics by inhalation easy and has removed the anesthetist and devices from the field of operation, so that the surgeon may operate without interference from difficulties arising from the administration of the anesthetic or the moving of the face mask. Surgical asepsis has been more easily maintained and the exposure of the operative field has been maximal with this method. When the operation is to be on the throat the tube may be passed by way of the nose (fig. 1), and when the operation is to be on the nose the tube may be passed by way of the mouth (fig. 2). In operations on the eye the tube may be introduced by either route. The intratracheal method is seldom necessary for a person who is to have an operation on the ear, although it is used to advantage in such an operation if there is any difficulty in establishing a free passage of air.

For the average patient, as preliminary medication $1\frac{1}{2}$ grain (0.09 Gm.) of pentobarbital sodium is administered by mouth the night

From the Section on Anesthesia, the Mayo Clinic.

This is a review article based on a lecture given at the meeting of the American Academy of Ophthalmology and Otolaryngology in New York on Sept. 29 and 30 and Oct. 1, 1936.

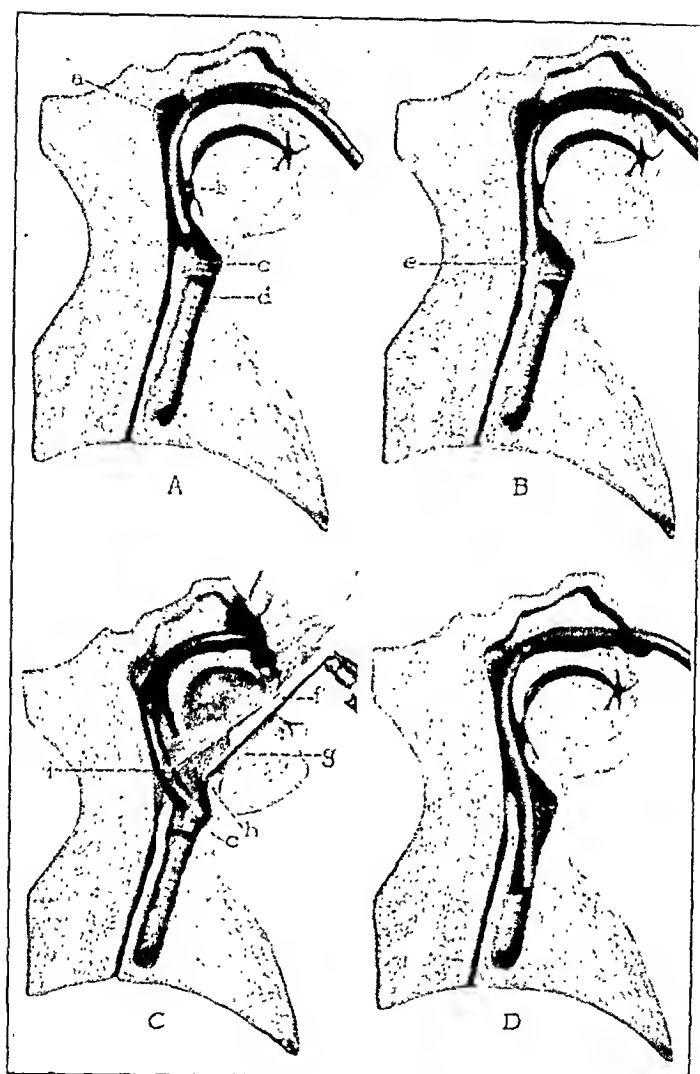


Fig. 1.—*A* shows the intratracheal tube inserted through the nose, passing through the nasopharynx (*a*), and extending deeply into the oropharynx (*b*), toward the larynx (*c*) and the trachea (*d*). *B* illustrates how in many cases the intratracheal tube enters the esophagus (*e*) instead of the trachea on the first attempt to insert it. If it continues to enter the esophagus with each of several attempts, the laryngoscope should be used. In *C* the laryngoscope (*f*) is inserted to depress the tongue (*g*) and the epiglottis (*h*), and the glottis (*c*) is visualized. The intratracheal tube may then be inserted through the glottis into the trachea by grasping the tip of it with the Magill forceps (*i*) and directing its course. Often, however, after the glottis is visualized the tube may be inserted by manual pressure on the tube from outside the nose. *D* shows the intratracheal tube in the proper position when it has been inserted through the nose.

before the operation.¹ The dose is repeated at 7 a. m. In the case of an average adult, $\frac{1}{6}$ grain (0.01 Gm.) of morphine sulfate and $\frac{1}{150}$ grain (0.0004 Gm.) of atropine sulfate are given by hypodermic injection at least thirty minutes before anesthetization is to be begun.

The essential points in using this intratracheal method are to spray the throat with a local anesthetic, such as a 5 or 10 per cent solution of butyn, and to induce anesthesia with a gas such as nitrous oxide, ethylene or cyclopropane.

A tube with an outside diameter such as will permit it comfortably to enter the nose, glottis and trachea is selected and lubricated with white petrolatum (figs. 3 and 4). When anesthesia has been established, the tube is inserted through the nose so that it follows the flow

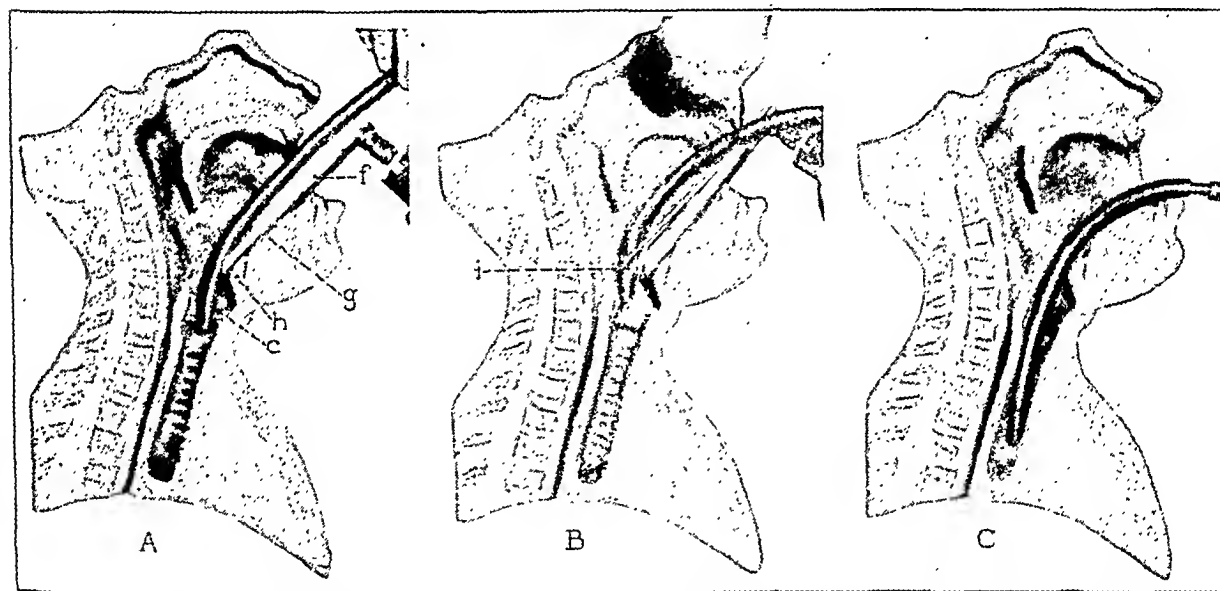


Fig. 2.—*A* shows oral intubation; *B*, oral intubation with a forceps, and *C*, the tube in place.

of air. A hollow blowing sound may then be heard, which assists materially in guiding the tube into the glottis. If the tube does not enter the glottis readily, the laryngoscope is used; the tongue and epiglottis are lifted, and the larynx is exposed. The tube may then be inserted from the outside by the use of Magill's forceps, the tip being guided between the tube and the vocal cords. In a rare case a divided airway may be used. It is inserted through the mouth, with the point against the glottis, and the intratracheal tube is inserted directly. The airway is removed a half at a time or may be allowed to remain in place.

1. Lundy, J. S.: *Rôle of Preliminary Medication in Anesthesia*, Surg., Gynec. & Obst. **63**:117-119 (July) 1936.

The advantages of the use of the tube are the effortless breathing of the patient and the easy administration by means of the gas machine of whatever mixture of anesthetics one wishes to use, including the administration of volatile agents by any device which will permit the



Fig. 3.—Four sizes of intratracheal tubes. The sizes from top to bottom are: (1) 8 mm. in diameter and 180 mm. in length; (2) 10 mm. in diameter and 215 mm. in length; (3) 11 mm. in diameter and 270 mm. in length, and (4) 14 mm. in diameter and 280 mm. in length.

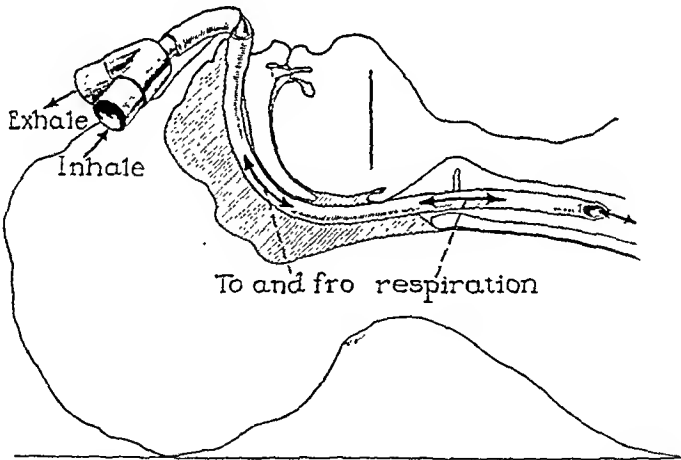


Fig. 4.—To avoid collapse of the tube, a metal connector is used when the tube otherwise would be forced into an acute angle, and the connector and adapter on the end of the inhaler tubing are joined by a short piece of rubber tubing. The direction of the inhaler tubing from the patient to the gas machine may be arranged at any angle convenient for the surgeon and the anesthetist by rotating the metal connector in that direction.

vapors to be inhaled or insufflated through the tube. Obviously, artificial respiration is an easy matter with the tube in place. The advantages are so apparent that it seems to me that one needs only to use

the method once or twice to realize them. This method is, I think, one of the outstanding advances in the means of inducing general anesthesia in the last ten years. One does not necessarily need a gas machine or mechanical devices to enjoy many of the benefits of the method. The tube may be inserted and left protruding slightly and fastened in place with adhesive tape. Then ether may be administered by the drop method so long as the end of the tube is kept under the mask.

Among the various agents used, some new ones have recently been employed. One is divinyl ether, which I have found less satisfactory than diethyl ether, although in the recent literature it has been discussed as being possibly and probably advantageous for use for myringotomy and for short operations when anesthesia is to be induced quickly and maintained for a short time. Because breathing has not been quiet and effortless with this agent, I have limited its use in my hands. Nitrous oxide is an extremely valuable agent, as it may be used with relative safety in cases in which cautery and diathermy are necessary. Ethylene has not been as popular, mostly because of its inflammability, although more oxygen can be given with it than with nitrous oxide. Cyclopropane, which was developed by Waters,² can be used with equal success in operations on the ear, but it too is inflammable and explosive, so that its use is limited in many of the operations on the eye, nose and throat if diathermy or cautery is to be used. It is, however, an excellent anesthetic agent when the conditions of operation permit its use, especially if one wishes to avoid the use of ether. Anesthesia may be induced, the intratracheal tube may be placed, and anesthetization then may be carried on with nitrous oxide and oxygen. However, there is some danger in having an inflammable gas on a gas machine, as the valve on the cylinder may not always be closed tightly and some gas may leak through into the mixture in the bag without the knowledge of the anesthetist or the surgeon until it becomes ignited. So, for the most part, when ethylene and cyclopropane are used, the cylinders should be on the gas machine only for the period during which the gas is used. This precaution may seem unnecessary, but the hazard is real enough to warrant it. The penalty of an explosion is too great if such care is not taken.

When cyclopropane is to be used, a special gas machine that has valves that are unaffected by the gas and one that includes a soda lime absorber for carbon dioxide is necessary. With this method, although the gas is expensive per cylinder it is not expensive per case. Obviously, the expense of nitrous oxide and of ethylene is equally reduced by the

2. Waters, R. M., and Schmidt, E. R.: Cyclopropane Anesthesia, J. A. M. A. **103**:975-983 (Sept. 29) 1934.

carbon dioxide absorption method, which was also developed by Waters.³ A warning in the use of cyclopropane is that the patient may easily become overdosed with it when anesthesia is too suddenly induced. The character of the pulse should be watched as deep anesthesia is approached, and if the quality of the pulse changes an increasing dose of ether should be added rather than increasing the concentration of gas in the mixture. While death has occurred from the too bold use of cyclopropane, I am satisfied that the lives of many patients have been prolonged by its use when disease of the lung has existed and when without cyclopropane ether must have been used, with subsequent exacerbation of a pulmonary disease.

For certain short operations, such as enucleation of an eye and myringotomy, one may induce and maintain anesthesia by the intravenous use of evipal (sodium *n*-methylcyclohexenylmethylmalonyl urea)⁴

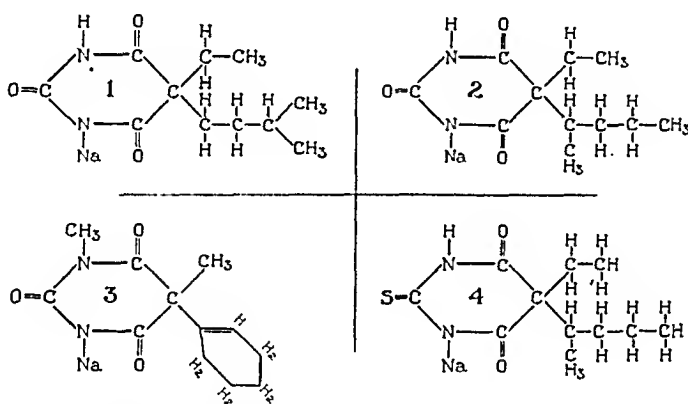


Fig. 5.—The chemical structures of (1) sodium amytal, (2) pentobarbital sodium, (3) evipal sodium and (4) pentothal sodium. Sodium amytal and pentobarbital sodium are similar in chemical structure, and they have a similar effect. Evipal sodium and pentothal sodium, although differing somewhat in chemical structure, have a similar effect. The one difference between the latter two substances and the former two substances is the utilization of bonds on the urea side of the barbituric acid nucleus as well as on the malonic acid side for substituents in the formulas for evipal sodium and pentothal sodium, while in the formulas for sodium amytal and pentobarbital sodium the bonds on the malonic acid side only have been utilized.

or of pentothal sodium (sodium ethyl 1-methyl butyl thiobarbituric acid).⁵ Since evipal has been sufficiently described in medical litera-

3. Waters, R. M.: Clinical Scope and Utility of Carbon Dioxide Filtration in Inhalation Anesthesia, *Anesth. & Analg.* **3**:20-22 (Feb.) 1924.

4. Evipal has not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

5. Lundy, J. S.: Intravenous Anesthesia: Preliminary Report of the Use of the Two New Barbiturates, *Proc. Staff Meet., Mayo Clin.* **10**:536-543 (Aug. 21) 1935. Pentothal sodium has not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

ture in this country and abroad, my remarks will concern pentothal sodium. Its relation to amytal (iso-amylethylmalonyl urea) and evipal is illustrated in figure 5. Pentothal sodium is from 30 to 50 per cent more potent than evipal. It is to be used only in a 5 per cent concentration. A respiratory stimulant may be mixed in the solution; 1 cc. of pyridine betacarboxylic acid diethylamine, 3 mg. of picrotoxin,⁶ or 3 grains (0.2 Gm.) of metrazol is added to 1 Gm. of pentothal sodium. The drug is given intermittently, as indicated in figure 6. Whenever possible, a "cotton butterfly" ⁷ is used over the nostrils or lips of the patient to indicate the inspiration or expiration of air, and the respirations should at no time be entirely inhibited by the drug.

The preliminary medication is the same as that used with other anesthetics.

The patient is asked to count, and it is best to induce anesthesia slowly enough so that the patient counts to between 20 and 30. Anesthesia may be easily induced in ten seconds, but there is a danger which is out of proportion to that of induction in from twenty to thirty seconds. I feel that the responsible person who uses this drug should never allow respiration to be stopped. Anesthesia should be induced slowly, and small doses should be given intermittently, when and if they are needed, the principle of the intermittent method of administering ether by the drop method being followed. The use of this method is on the increase, and the procedure is one that needs to be mastered by those who are interested in it. It is not a method that should be used by those who are inexperienced or who are not cautious. It is not recommended in cases in which the patient is a child less than 10 years of age because of the small caliber of the respiratory passages, which, together with the depression of respiratory action by the drug, makes pulmonary ventilation by the patient difficult. Cyanosis of the patient should be avoided, and if it occurs it should be treated promptly, as the outcome may easily be untoward if the cyanosis persists.

The use of pentothal sodium should be avoided in cases in which the patient suffers from respiratory disease, one of the symptoms of which is dyspnea when the patient is at rest. Its use for manipulations in the throat is usually not satisfactory unless the throat has been anesthetized by a local anesthetic before general anesthesia has been induced for bronchoscopy and other such procedures. If the intravenous anesthetic is to be used for tonsillectomy, the intratracheal tube should be inserted so that there will be a free airway throughout the operation and

6. Picrotoxin has not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

7. Lundy, J. S.: A Method of Minimizing Respiratory Depression When Using Soluble Barbiturates Intravenously, *Proc. Staff Meet., Mayo Clin.* **10**:791-792 (Dec. 11) 1935.

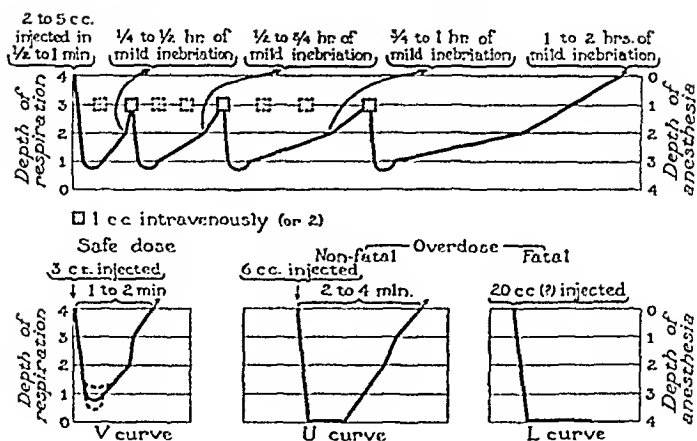


Fig. 6.—Diagrams showing the effect of the doses of pentothal sodium given by intravenous injection for a man aged 45, weighing 160 pounds (72.6 Kg.) and 6 feet (182.9 cm.) tall. The preliminary medication consisted of the administration of $\frac{1}{6}$ grain (0.01 Gm.) of morphine and $\frac{1}{150}$ grain (0.0004 Gm.) of atropine given by hypodermic injection and the oral administration of 1.5 grain (0.09 Gm.) of pentobarbital sodium. The effect produced by the administration of a 5 per cent solution of pentothal sodium by different technics is shown. To avoid the necessity of drawing two curves in each diagram it is assumed that anesthesia deepens as respiration becomes more shallow and that this relationship is exact. The relationship may not be exact, but for diagrammatic purposes the assumption will serve. The top diagram shows the effect on the depth of respiration and of the anesthesia produced by repeated safe doses. After the first dose, respiration becomes more shallow and anesthesia deeper for a time; then, as the patient begins to recover, respiration deepens and anesthesia becomes lighter until the patient reaches a stage of inebriation which will last for from a quarter of an hour to half an hour. If the operation has been completed in the relatively short period of anesthesia conferred by the first dose, no more of the drug need be administered, and after the stage of inebriation has passed recovery is complete. However, if a longer period of anesthesia is required to permit the operation to proceed, a second dose is administered, and so on, with the effects on respiration, anesthesia and duration of inebriation that are represented in the curve. When similar successive safe doses are administered, the last one often, but not always, has a longer effect than the previous ones. The dose varies according to the patient's resistance to the drug. One person may require 2 cc. of a 5 per cent solution for each cubic centimeter, as indicated in the top curve, at least for the first two or three injections. The diagrams marked V curve, U curve and L curve, which show the vulnerability for a 5 per cent solution of pentothal sodium, illustrate the relative effect of the safe dose and of the overdose. The V curve illustrates the safe dose for the short operation, for example, manipulation of a stiff wrist joint. The U curve illustrates the nonfatal result of a moderate overdose; respiration is practically completely depressed for a short time, and recovery is prompt if no more of the drug is injected. It shows that the period of anesthesia is longer than it is when a small dose is injected, and this period of anesthesia is coincidental with respiratory depression. The effect of an overdose, that is, of a dose large enough to be fatal, is illustrated in the L curve and needs no further explanation. Certain modifications of this technic might be carried out, and the L curve could be broken down into two U curves, the second of which might possibly illustrate a fatal result, based on the results obtained in the top curve or if the condition is complicated by any respiratory obstruction.

so that the danger of aspiration of material from the throat will be minimized.

Certain advantages of the method, such as the portability of the agent, the fire-proof conditions, the quick induction of anesthesia, the short period before recovery, the absence of excitement in most cases and also a minimum of nausea and vomiting, are obvious. There is evidence that this drug, if used by this method, tends to delay the onset of shock—a valuable consideration in certain cases in which the patient is debilitated. It usually lowers the blood pressure in cases of hypertension and may be used to advantage in such cases if it is otherwise indicated. The use of pentothal sodium is described elsewhere, and for the benefit of those who are familiar with evipal it may be stated that its use is similar to that of the latter drug, except that a smaller dose of pentothal sodium must be used.

The rectal method of anesthesia was formerly carried out with olive oil and ether and later with tribromethanol in amylene hydrate (avertin fluid).⁸ The use of the latter agent in a dose of from 80 to 90 mg. per kilogram of body weight, so that its effect is only that of basic anesthesia, has been satisfactory. It permits operation under minimal additional amounts of local or inhalation anesthetics. Its use in doses sufficient to produce anesthesia is not to be recommended in most instances.

The rectal use of short-acting barbiturates is in the experimental stage and need not be discussed further as yet.⁹

The use of any anesthetic agent or method should be backed by a sufficiently good reason so that its use can be justified in the event of any untoward result. Carelessness in the selection and application of these methods, especially if the routine use of one or more is attempted, may easily lead to difficulties. For example, in using the intravenous method one should master venipuncture if satisfaction is to be expected. It is necessary to know that a needle should always be selected which one feels will easily enter the vein. If the needle is somewhat large, the bevel should be laid against the skin rather than be turned away from it; then when the proximal wall of the vein has been entered there will be a minimal chance that the needle will perforate the distal wall.

I would recommend that the surgeon review the question of modern anesthesia and see the newer methods demonstrated in competent hands, just as one must observe the technical steps of an operation in order clearly to visualize the procedures.

8. Avertin has not been accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

9. Gwathmey, J. T.: Rectal Administration of Evipal Soluble: A Safe, Reversible and Controllable Preanesthetic Medication: a Preliminary Report, *Am. J. Surg.* **32**:411-416 (June) 1936.

News and Notes

UNIVERSITY NEWS

Dr. Francis Heed Adler has been appointed to the chair of ophthalmology at the University of Pennsylvania, as successor to the late Dr. T. B. Holloway.

GENERAL NEWS

The National Eye Service.—A new central clinic of the National Eye Clinic has been established at Castle Street, Torquay, England. At the opening ceremony Mr. A. W. Ormond, consulting ophthalmic surgeon to Guy's Hospital, referred to the inauguration of the National Eye Service by the British Medical Association as a public service designed to provide a thorough examination of the eyes by a competent ophthalmic medical practitioner and any necessary glasses at a cost within the means of those who cannot afford ordinary fees but who deserve the best ophthalmic treatment.

SOCIETY NEWS

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom will be held in London on April 29 and 30 and May 1, 1937.

The subject for discussion will be "The Rarer Forms of Keratitis." The following members will open discussions: Mr. F. A. Williamson-Noble (neurologic aspects), Mr. Affleck Greeves and Mr. J. Doggart (clinical aspects), Mr. Harrison Butler (slit-lamp aspects), Mr. Frank Law (ultraviolet light therapy) and Dr. Douglas Webster (roentgen therapy). Other members wishing to take part in the discussion should notify Mr. H. B. Stallard, 35 Harley Street, London, W. 1, before February 28.

Further information regarding the program and the presentation of papers may be secured from Mr. Stallard.

Philadelphia County Medical Society, Eye Section.—The monthly meeting of the Eye Section of the Philadelphia County Medical Society was held on December 3. The following program was presented: teaching conferences, by Drs. Lillie, Cowan and Lehrfeld; presentations of clinical cases from the Wills Hospital, by Dr. W. D. Angle, and a symposium on iridocyclitis and uveitis, in which were discussed "Etiology," by Dr. Leo F. McAndrews, "Diagnosis," by Dr. Leighton F. Appleman, and "Treatment," by Dr. Charles R. Heed.

Obituaries

THOMAS B. HOLLOWAY, M.D.

1872-1936

Thomas Beaver Holloway was born in Danville, Pa., on March 24, 1872. He died at his home in Merion Station, Pa., on Aug. 18, 1936. He was the son of S. William Kerling Holloway and Ruth Ann Holloway. Dr. Holloway was twice married. By his first wife, to whom he was married in 1902, Cordelia Gearhart Woolley of Danville, Pa., he had one son, Thomas B. Holloway Jr. In 1931 he married Florence Jane Laird of Philadelphia, who survives him.

His preliminary education, which was completed in 1888, was received at the Danville Academy. For a period of two years he was associated in business with his father. He then entered Lafayette College, and graduated with the degree of B.S. in 1894. In 1897 his alma mater conferred on him the degree of M.S., and in 1926, that of Sc.D.

On biologic qualifications he was admitted to the second year of the medical school of the University of Pennsylvania and graduated in 1897. After serving an internship for two years at the Philadelphia General Hospital, Dr. Holloway began the general practice of medicine in Philadelphia.

Dr. Holloway's career in ophthalmology began in 1902, when he was appointed assistant surgeon at the eye dispensary of the University Hospital in the medical school of the University of Pennsylvania. He was appointed instructor in ophthalmology in 1905, associate professor in 1923 and professor in 1924, succeeding G. E. de Schweinitz. His talents in the field of ophthalmology were quickly recognized, and his services were sought by a number of important hospitals and teaching institutions in and around Philadelphia. In 1914 he was elected professor of ophthalmology at the Philadelphia Polyclinic, and in 1918 was appointed professor of ophthalmology in the graduate school of medicine of the University of Pennsylvania. He was made vice dean for ophthalmology in the latter institution in 1920.

After entering private practice Dr. Holloway spent seven profitable years as the personal assistant of Dr. G. E. de Schweinitz, both in the de Schweinitz' office and at his clinic in the University Hospital. During this period it was only natural that an intimate professional and personal relationship should have developed between the two men.

Many of Dr. Holloway's earlier papers were, therefore, presented with Dr. de Schweinitz as co-author, and this happy custom was continued for some years. Among the most notable contributions emanating from this joint authorship were "Pulsating Exophthalmos," an essay based on sixty-nine case histories, "The Operative Treatment of



THOMAS B. HOLLOWAY
1872-1936

Papilloedema Dependent upon Increased Intracranial Tension" and "Certain Visual Defects in Hypophysis Disease."

Under his own authorship and, frequently, in association with other colleagues, Dr. Holloway presented before ophthalmologic societies or general medical societies well over one hundred interesting and impor-

tant communications, all of which have been published and preserved for posterity. As an extemporaneous speaker on medical subjects he spoke fluently and with conviction, invariably displaying an unusual knowledge of the current literature on the question under discussion.

The esteem in which Dr. Holloway was held by his associates resulted in his election to membership in numerous ophthalmologic, neurologic, pathologic and general medical societies in this country and abroad, and in many of these he was honored with positions of distinction and responsibility. From 1918 to 1925 he was secretary-treasurer of the American Ophthalmological Society and for a period of ten years edited the society's Transactions; he was president of the society in 1932.

In 1918 he was a member of the House of Delegates of the American Medical Association, and was chairman of the Section on Ophthalmology from 1929 to 1930. Elected to fellowship in the College of Physicians of Philadelphia in 1906, he served as clerk of the Section on Ophthalmology from 1909 to 1913 and became chairman of the section in 1924. He was a member of the John Morgan Society, the American Academy of Ophthalmology and Otolaryngology, the Société française d'ophtalmologie and the Ophthalmological Society of the United Kingdom, as well as numerous other special and general medical organizations.

Dr. Holloway had a large and influential private practice in addition to unusual opportunities for clinical and pathologic research in the general and special hospitals with which he was connected. As a clinician he was painstaking, thorough and skilful and never lost sight of the fact that the eye and its diseases were closely allied to general disease and to diseases of the central nervous system. He was an expert ophthalmoscopist and retained to a remarkable degree a clear mental picture of what he had observed in the fundus. His description of the departures from the normal were accurate, instructive and illuminating. He displayed sound surgical judgment and as an operator did his work with meticulous care. Never slow to adopt new surgical procedures if they had proved to be meritorious, he usually selected the type of operation which he himself did best, and his results compared favorably with those of larger surgical experience.

As a teacher he admirably succeeded in imparting his knowledge in a clear, convincing manner, emphasizing always the close relationship between ocular disease and constitutional disease and ever striving to impress on the undergraduate the importance of ocular examinations in the diagnosis of central lesions, cardiovascular disease and renal disease. As one colleague expressed it, "He continually sought to make

the students eye conscious, rather than endeavored to make of them eye practitioners"—a difficult undertaking at best but one in which his efforts were appreciated by the student body.

Any student of medical history could well record the achievements of Thomas B. Holloway in his special field of activity, but only his close personal friends can give a proper estimate of the outstanding characteristics of the man. Always an indefatigable worker and a diligent reader of ophthalmic literature, he found little time for relaxation, and the demands of social life had little appeal for him. When, however, he was able temporarily to cast aside the burdens and responsibilities incident to his work and surround himself with a group of congenial friends there was never a more delightful companion or one who entered into the spirit of the occasion with more enthusiasm. He took great delight in playing practical jokes on his intimate friends and had a keen sense of humor and a cheerful, buoyant spirit which ever shed its influence on those around him. He was a kind and indulgent father, a devoted and sympathetic husband and a true friend. No task was too hard, no effort too great, when his help and cooperation were sought by friends or professional associates. While a member of several clubs, he was rarely able, because of his active professional life, to use them except for the entertainment of his friends. After the day's work was done he preferred the quiet of his home, where he could be surrounded by his family and, not infrequently, by his close associates.

I had the privilege of knowing Tom Holloway intimately for a long period and saw him frequently during his active years and throughout his last illness. This friendship, which will ever be a cherished possession, endured until the end. Any appraisal of his life, filled as it was with great professional distinction, high honors and the esteem of his fellowmen, would not be complete without reference to the courage he displayed during times of adversity. He had reverses and at times faced conditions which "try men's souls," but never did they embitter him or alter the tenor of his life. Fully aware for several months that his last illness would terminate fatally, he faced death, "the last enemy to be destroyed," with calmness and a courage which excited the admiration of those who were privileged to be with him in those trying hours. Only eager to be spared long enough to "put his house in order" and to dispose of some of his most cherished possessions, he complacently accepted the inevitable outcome without fear or resentment and in his last days must have derived consolation from the thought that he had fought a good fight and had kept the faith.

HUNTER H. MCGUIRE.

The friends of Dr. Holloway little thought that the illness which befell him in December 1935 would suddenly cut short his life's work and soon after cause his death.

Dr. Holloway's success was assured through his love of work and the high degree of intelligence which he brought to it. The success of the department of ophthalmology in the Graduate School of Medicine of the University of Pennsylvania was largely due to his enthusiastic efforts. He planned the curriculum on a broad basis and in time shaped it into a course which has made it outstanding. In his teaching in the undergraduate school he endeavored to make the students eye conscious rather than to make of them eye practitioners.

In his death the American Ophthalmological Society has lost one of its most devoted members. It claimed much of his time and thought. He was deeply interested in the future of the younger ophthalmologists about him, and to them his death will be a great loss. He will be greatly missed in the Section on Ophthalmology of the College of Physicians of Philadelphia, where he was regular in his attendance, presented many of his best papers and participated freely in discussions.

He was a diligent reader of ophthalmic literature and had a retentive memory, so that even in his extemporaneous discussions he could quote the literature on a subject with amazing facility.

He disliked publicity, was reserved about his personal affairs and was strong in adversity. Reverses did not embitter him or much alter the tenor of his life. He had a cheerful, buoyant disposition.

Ophthalmology has lost a devoted practitioner; students, an enthusiastic teacher, and his colleagues, a steadfast friend.

Deep sympathy is felt for his widow and his son.

W. ZENTMAYER.

The loss of Tom Holloway will be keenly felt by a large circle of acquaintances, while to his many friends his death leaves a void impossible to fill. Especially will his absence be noted by the several ophthalmologic societies of which he was a most active member. Possessed of a most retentive memory of the literature as well as of the contributions of various authors, he was never at a loss for opinions to confirm his own judgment, which was founded on a thorough knowledge and careful study of his subjects. A good judge of men, he was rather outspoken in criticism, but generous in praise, as the case might be.

He was a man of great uprightness and, once having conceived his duty, could not be swayed from his course. He was slow to make friends, but once his friendship was given it was never retracted. He was kindly, considerate, generous to a fault and invariably a gentleman.

He was a great lover of the outdoors and an ardent angler ; nothing delighted him more than to follow the course of a stream where trout or salmon were to be found.

Although he was extremely reticent, there were those who were privileged to know him well ; these were aware of his high ideal of service and appreciated that it was based on a deep religious conviction.

So Tom Holloway played the game of life as, in years gone by, he had played the game of baseball, taking defeats and victories in his stride, and on the realization of the near approach of the end of his earthly tenure he set about placing in order his material affairs without the least shadow of doubt in his heart.

LOUIS S. GREENE.

CLARENCE KING, M.D.

1877-1936

The many friends of Clarence King were shocked to hear of his sudden death while he was bathing in Grape Bay, Paget, Bermuda, on August 15. The abrupt ending of this useful life while Dr. King was apparently in perfect health and enjoying full activity, at an age so far in advance of the limit of human life set by the Psalmist and constantly demonstrated by human experience, accentuates the keenness of the regret of ophthalmologists that one so favored by nature should be taken from the eminent position he had earned and adorned.

Dr. King was born in Newport, Ky. He graduated from the Medical College of Ohio in 1901 and served his internship at the Good Samaritan Hospital, Cincinnati. Later he studied ophthalmology in Vienna and Berlin, returning to Cincinnati to practice his specialty.

In 1911 he went to Amritsar, India, where he operated in the clinic of Colonel Henry Smith. During the war he served in the Medical Corps in France and in Coblenz, Germany. After he returned home he was in charge of the eye service of the Cincinnati General Hospital and also of the Children's Hospital. He served as assistant professor of ophthalmology under Prof. Victor Ray on the faculty of the University of Cincinnati College of Medicine, and became professor when Dr. Ray died. These three positions occupied a great deal of his time and thought, and the increasing demands made everywhere today on teachers in medical schools were met by him and satisfied.

He visited Vienna in 1927 and studied especially the effects of the treatment of tuberculosis of the eye with tuberculin.

Dr. King was prominent in the Cincinnati Academy of Medicine, the Ohio State Medical Society, the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology and the Section on Ophthalmology of the American Medical Association. He was a faithful attendant at the meetings of these societies, taking an active part in the discussion and contributing papers of decided merit.

His last paper was read before the American Ophthalmological Society in June 1936.¹ A few years ago he contributed an article on heterochromia,² which was the first effort in this country to separate

1. King, C.: Chronic Subdural Hematoma as a Cause of Choked Disk, *Arch. Ophth.* **16**:903 (Nov.) 1936.

2. King, C.: *Tr. Am. Ophth. Soc.* **14**:339, 1927.

the group of conditions included under that title. Another important contribution dealt with the use of tuberculin in the various forms of tuberculosis of the eye.³

I have suggested Dr. King's personal charm, innate courtesy and kindness. Perhaps these will be better explained to those who had not the good fortune to know him by saying that the tribute of Halleck to his friend Drake, "None knew thee but to love thee nor named thee but to praise," can be appreciated fully by those who knew Clarence King.

R. I. LLOYD.

3. King, C.: Therapeutic Value of Tuberculin in Ophthalmology, *Arch. Ophth.* 1:713 (June) 1929.

1864—1936

Robert Henry Elliot, the son of a colonel in the army, studied medicine at St. Bartholomew's Hospital, from which he graduated brilliantly with many honors. After entering the Indian Medical Service in 1892, he obtained civil employment in Madras in 1894 and worked in various capacities until he became, in 1904, the superintendent of the Government Ophthalmic Hospital in Madras and professor of ophthalmology in the Madras Medical School. He was instrumental in raising this hospital to a leading position in the study and treatment of diseases of the eye. Here he had the opportunity of making his name world known in his chosen specialty, particularly in connection with the operation for sclerocorneal trephining in chronic glaucoma.

In 1914 he returned to England on account of ill health and started an active practice in London. He continued his literary activities, became lecturer on ophthalmoscopy at the London School of Hygiene and Tropical Medicine and delivered the Hunterian Lecture for 1916-1917 before the Royal College of Surgeons. While in London, as a member of the Council of the British Medical Association he was instrumental in bringing about many needed reforms in the Indian Medical Service. In addition to being an indefatigable worker, Elliot wrote with great facility and clarity. Among the many books from his pen "Glaucoma" and "Tropical Ophthalmology" are best known.

The operation of trephining in chronic glaucoma, which is practiced all over the world and is known as the Elliot operation, did much to remedy that dreaded disease and save many sufferers from blindness. According to Kirkpatrick,¹ Elliot "recognizing the value of Lagrange's work on this subject set himself to devise a safer method of securing a filtering cicatrix, and evolved the idea of removing subconjunctivally with a trephine, a disc of the sclero-corneal tissue." The success which this operation had in checking the progress of chronic glaucoma gave a renewed impetus to the study of this disease, and in Elliot's "A Treatise on Glaucoma" the reader will find the subject treated in a complete and scientific manner.

Elliot visited the United States in 1913 and demonstrated his operative technic in various ophthalmic hospitals. He contributed a number of articles to the ARCHIVES, the last on "Trephining,"² in which he

1. Kirkpatrick, Henry: Brit. M. J. 2:1062 (Nov. 21) 1936.

2. Elliot, R. H.: Trephining, Arch. Ophth. 8:797 (Dec.) 1932.

emphasized various points in this operation which make for success and which bear constant repetition.

At the Seventeenth International Congress of Medicine in 1913 he was a reporter on the subject of glaucoma.

Elliot's interests, outside of ophthalmology, were snakes, to which he had devoted much study, and magic; a remarkable book by him on the latter subject, "Myth of the Mystic East," appeared in 1934.

His wife, who had been a great help to him in his scientific investigations and writings, died eleven years ago. Three sons survive.

ARNOLD KNAPP.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

FORMATION OF SYNECHIAE BETWEEN THE MARGIN OF THE OPTIC CUP AND THE LENS IN THE EMBRYO. T. A. Vos, *Klin. Monatsbl. f. Augenh.* 96: 452 (April) 1936.

Two intra-ocular formations occurring in microphthalmos are described: a membrane between the ciliary body and the posterior surface of the lens, and a similar membrane connecting the retina and the lens. Eight selected cases are reported and well illustrated. Vos found that the membrane between the lens and the inner surface of the eyeball consisted either of a predominantly uveal or a predominantly retinal derivative. The mechanism is represented by an embryonal synechia between the lens and the margin of the optic cup. Isolated degeneration of the latter is considered the cause of this formation of a synechia. In this connection an eyeball is described which presented a congenital connection between the lens and a posterior portion of the retina. Vos compares this connection with a formation of folds described by several authors as *ablatio falciformis* of the retina. Incidental findings were colobomas, fibrous "apparent" lenses, cysts of the iris and circular detachment of the retina with the optic nerve drawn inside the eyeball. Vos considers it possible that the presence of the synechia may form an opportunity for avoidance of the hyaloid artery, leading to its preservation.

K. L. STOLL.

THE ANATOMY OF ZINN'S RING. A. VILA-CORO, *Klin. Monatsbl. f. Augenh.* 96: 477 (April) 1936.

Vila-Cora proves by his research and by detailed anatomic description that Zinn's ring is a slit. He suggests that one ought to term the passage from the orbit into the interior of the skull not "Zinn's ring," but "Zinn's canal" or, still better, "Zinn's slit."

K. L. STOLL.

Bacteriology and Serology

POSTOPERATIVE ENDOGENOUS INFECTIONS OF THE EYE. D. KRAVITZ, *Am. J. Ophth.* 19: 328 (April) 1936.

Kravitz reports the extraction of a cataract in which delivery of the lens was very difficult, as though it were held down below, and when delivered it showed a small amount of soft whitish exudate at its lower pole. Two days later there was pus in the anterior chamber, and a smear showed numerous extracellular and intracellular organisms, which the pathologist diagnosed as gonococci. On being questioned,

the patient finally recalled a gonorrheal urethritis that occurred 60 years before, when he was aged 18. The infection continued, and the eye finally became phthisical. A complement fixation test was negative. He quotes various authors and speculates on the possibility of these infections lying dormant for long periods.

W. S. REESE.

Biochemistry

THE CIRCULATION OF BLOOD IN THE EYE AND VITAMIN C. W. BUSCHKE, *Arch. f. Augenh.* 109: 691, 1936.

The amount of cevitic acid in the aqueous depends on the amount of the oxidized form of vitamin C in the blood. The vitamin C coming from the blood is reduced by the lens to cevitic acid. This acid cannot get back into the blood stream because normally the blood-aqueous barrier is impermeable to its passage. If, however, as numerous experiments have proved, this barrier is made more permeable, as by subconjunctival injections of salt solution, the amount of cevitic acid in the aqueous falls. Further, if the vitamin C in the blood is increased by feeding or by intravenous injection of a synthetic preparation of vitamin C, the cevitic acid in the aqueous rises. This demonstrates the interesting fact that the lens not only can reduce ordinary amounts of vitamin C presented to it normally but has the capacity to take care of considerably larger quantities when these occur under experimental conditions.

Three factors are of importance in determining the quantity of cevitic acid in the aqueous: (1) the size and blood flow of the uveal capillaries, (2) the permeability of the capillary walls for vitamin C and (3) their permeability for cevitic acid.

The third factor was investigated in previous papers (Goldmann and Buschke: *Arch. f. Augenh.* 109: 314, 1935). The present communication deals with variations in the first two factors. These two factors cannot at present be separated. The authors therefore elected to use drugs which increase and decrease the size and the permeability of the capillaries of the uveal tract, i. e., acetylcholine and epinephrine.

In a series of rabbits a subconjunctival injection of epinephrine was given in one eye, followed at regular intervals by drops of epinephrine in the conjunctival sac, and 100 mg. of a synthetic preparation of vitamin C was given intravenously to raise the vitamin C content of the blood. The cevitic acid content of each of the eyes was then determined. As was to be expected, the control eye contained much more cevitic acid than the eye treated with epinephrine. They ascribe this effect to diminution in the supply of vitamin C reaching the anterior chamber of the treated eye—hence, less for the lens to convert into cevitic acid. They do not decide whether the widely dilated pupil in the eye treated with epinephrine was a factor in producing this effect or not.

In a second series of rabbits acetylcholine was injected subconjunctivally in one eye, and at varying periods the cevitic acid contained in each anterior chamber was determined. In the majority of the experiments a considerable increase in the cevitic acid content of

the eye treated with acetylcholine was found, but the effect was not constant, and in one case a lower content of cevitic acid was found. There seemed to be a more or less general agreement between the increase in cevitic acid and the degree to which the capillaries retained their normal impermeability, for when the protein in the aqueous was found to be high after the use of acetylcholine (this was used as an index of increased permeability of the uveal capillaries) the cevitic acid was not increased. Acetylcholine, according to these authors, must therefore have some peculiar effect on the eye which leads to an increase in cevitic acid, besides its effect in increasing the permeability of the walls of the blood vessels. This action may be an increase in the blood flow through the uveal capillaries, which brings a greater amount of vitamin C into the anterior chamber. Here it is changed into cevitic acid, and in this form it cannot escape from the anterior chamber back into the blood stream.

A discussion of technical points follows to prove that the effects the authors observed are in reality due to changes in the intra-ocular circulation.

F. H. ADLER.

Color Sense

ANOXEMIC COLOR FATIGUE: A LATENT DISTURBANCE OF THE COLOR SENSE. K. VELHAGEN, *Arch. f. Augenh.* 109: 605, 1936.

A group of persons possessing normal color vision were subjected to reduced barometric pressures in a pressure chamber and their color vision tested with the anomaloscope. Readings were taken at pressures corresponding to sea level, 3,000, 5,000 and 6,000 meters of altitude. The effect of lowered oxygen tension at normal barometric pressure was not investigated.

It was found that a number of persons show no change in their color perception at high altitudes, but that a greater number show a considerable reduction in their discrimination for colors and brightness. In some persons who have normal color vision at normal barometric pressure a definite disturbance in color vision develops which simulates that of a congenital anomalous trichromat. Further, anomalous trichromats can be changed to dichromats by reducing the barometric pressure.

The significance of these changes is discussed, and their importance in aviation is pointed out.

F. H. ADLER.

Cornea and Sclera

TATTOOING OF CORNEAL OPACITY WITH GOLD AND PLATINUM CHLORIDE. J. N. DUGGAN and B. P. NANAVATI, *Brit. J. Ophth.* 20: 419 (July) 1936.

In twenty-nine cases corneal opacities were tattooed with gold chloride. The following summary is substantially that given by the authors:

In twenty-five cases the opacities were tattooed with gold chloride and tannin, and in four, with gold chloride and hydrazine hydrate, with fairly good cosmetic results. Tattooing with tannin, which produced

a brown tint, was more painful than that with hydrazine hydrate, which gave a black color.

A slightly acid 4 or 5 per cent solution of gold chloride was applied to the denuded surface of the leukoma for four or five minutes. The strength of the tannin was 1 per cent, while that of the hydrazine hydrate was 2 per cent.

In vascular leukoma the color obtained in tattooing by either method slightly faded and partly disappeared in some cases, in which a second operation was required.

In fourteen cases the opacities were tattooed with a freshly prepared 2 per cent solution of platinum chloride and a 2 per cent solution of hydrazine hydrate. The color obtained was jet black.

Iridectomy could be done either before or after tattooing by either method.

Of the cases reported in this article, two are of special interest. In one, an opacity which was tattooed with gold chloride and tannin showed a fairly good cosmetic effect even after two years, while in the other case, in which an opacity was tattooed with platinum chloride and hydrazine hydrate, the slightly faded black color had remained unchanged for five years.

W. ZENTMAYER.

A CASE OF SCLEROSING KERATITIS PROFUNDA. A. VISWALINGAM, Brit. J. Ophth. 20: 449 (Aug.) 1936.

The patient was a man aged 59 who had had renal calculi. The corneal condition was symmetrical, involving both eyes, and was accompanied by attacks of intermittent bulbar congestion lasting from twenty-four to forty-eight hours. With the attacks of congestion, calcium oxalate crystals were found in the urine. Later attacks of secondary glaucoma supervened. The left eye became blind and painful and was enucleated. A detailed pathologic report on the eye is given by Stallard. The corneal lesion had predominant inflammatory characters; such degenerative changes as were present were probably secondary to these. The article is illustrated.

W. ZENTMAYER.

SPECTROGRAPHIC STUDY OF THE KAYSER-FLEISCHER CORNEAL RING. A. POLICARD, P. BONNET and G. BONAMOUR, Compt. rend. Soc. de biol. 122: 1120, 1936.

The greenish ring of the cornea characteristic of Wilson's disease (hepatolenticular degeneration) was submitted to histospectrographic study according to Policard's special technic. Copper was noted in an appreciable amount, but no silver, iron or zinc. The copper of the corneal ring is undoubtedly associated with the sclerotic liver observed in patients with this disease.

J. E. LEBENSOHN.

Experimental Pathology

THE SERUM LIPASE IN NAPHTHALENE CATARACT. D. MICHAIL and I. PACURARIU, Compt. rend. Soc. de biol. 122: 1125, 1936.

In rabbits given naphthalene the lipase of the blood serum diminishes, reaching its lowest point when the opacity of the lens becomes

total. Thereafter the lipase rises slowly but does not again attain its normal level for from four to five months. The lipase of the lens and retina is probably similarly affected. This suggests that a pancreatic disease affecting the production of lipase may be at the basis of certain senile cataracts, especially when these are associated with diabetes.

J. E. LEBENSOHN.

General

OPHTHALMOSCOPIC SIGNS OF DEATH. C. R. SALSURY and G. S. MELVIN, *Brit. M. J.* 1:1249 (June 20) 1936.

Salsbury and Melvin have established by clinical and experimental observations that in life the movement of the column of blood in the retinal veins is visible. As death approaches, the stream becomes slightly irregular and lumpy. Later (with cessation of cardiac activity) there is fragmentation of the column, the masses of blood moving toward the optic disk and then dropping over the edge of the optic cup. This movement is visible for from ten to twelve minutes. After all movement of the blood has ceased, the interrupted columns are visible as long as the media remain transparent.

This movement of blood in the veins is ascribed to an equalization of pressure between the arteries and veins, due to isolated arterial contractions.

This test has frequently been applied to patients in profound shock, but always with negative results. No patient in whom this phenomenon has been observed has subsequently shown the slightest evidence of life.

The conclusions are: 1. If the retina appears as in life, the heart is still functioning, and artificial respiration is indicated. 2. If this movement of the blood in the veins is visible, the heart has stopped for not more than fifteen minutes; attempts at resuscitation are indicated, but the prognosis is bad. 3. If fragmentation is visible and all movement of the venous blood has ceased, recovery is probably impossible.

W. F. DUGGAN.

BIOLOGIC ACTION OF DIFFERENT FORMS OF RADIANT ENERGY ON OCULAR TISSUES. G. LEPLAT, *Ann. d'ocul.* 173:433 (June) 1936.

Leplat refers to the fact that Strohl a little over a year ago dealt with this subject before the Ophthalmological Society of Paris.

Besides roentgen rays and the radiations of radioactive bodies, other forms of radiant energy interest ophthalmologists either because they are used in therapeutics or because they are the cause of ocular lesions the mechanism of which is still in controversy. The question of so important a matter as glassblowers' cataract and of the action of infra-red rays is still much debated.

The author's subject is divided into: the different forms of radiant energy and the conditions of their action; the absorption of radiant energy by the ocular tissues; the action of radiant energy on the ocular tissues; the action of infra-red rays; the effects of heat and the action of ultraviolet rays and the action of roentgen rays and of radium on the ocular tissues.

S. H. McKEE.

General Pathology

A METHOD TO KEEP EXPLANTED LENSES ALIVE. A. BAKKER, Arch. f. Ophth. 135: 581 (July) 1936.

The work reported in this paper was done in the physiologic institute of the University of Gröningen, and a method devised by the director of this institute, de Haan, was used. The lenses of adult rabbits were removed under sterile conditions and transferred to shallow water-tight, air-tight glass chambers which permitted microscopic examination through their walls. A slow but constant stream of a nutritive fluid was led through each chamber. As the nutritive fluid, Bakker used the abdominal fluid which rabbits produce on intraperitoneal injection of large amounts (from 750 to 1,000 cc.) of Ringer's solution. The composition of this fluid, which really is a very dilute exudate, resembles that of normal aqueous. Before it entered the glass chambers carrying the lenses, it was saturated with air. Its gas content after it had passed through a chamber permitted calculation of the consumption of oxygen by the lens. Under the conditions of these experiments, the consumption of oxygen and dextrose, the macroscopic and microscopic appearance and the response to injuries of the capsule of the lens of the mature rabbit remained normal for several days, which facts indicate that the lens was kept alive.

P. C. KRONFELD.

Glaucoma

RELATIONS BETWEEN OBSTRUCTION OF THE CENTRAL RETINAL VEIN AND GLAUCOMA. J. KRAUSE, Arch. f. Ophth. 135: 173 (March) 1936.

Krause reviews the work of Harms, Coats, Verhoeff, Scheerer, von Hippel and Kiel and Salzmann on primary glaucoma leading to obstruction of the central vein and on obstruction of the central vein leading to secondary glaucoma. Krause himself has studied anatomically twenty eyes which were enucleated because of absolute glaucoma. Only sixteen of these eyes exhibited pathologic changes of the central retinal vein. The vein was always greatly narrowed; often its wall was indented and the lumen filled with endothelial cells, fibrocytes and a network of fibers. A true thrombosis could not be diagnosed with certainty in a single case. Krause's observations were not essentially different from those of the aforementioned authors. He discusses a number of well known factors, and also a few new ones which predispose the intralaminar portion of the retinal vein to obstruction.

P. C. KRONFELD.

PROVOCATIVE TESTS FOR GLAUCOMA: THE INTERMITTENT GLAUCOMA. G. OHM, Arch. f. Ophth. 135: 537 (July) 1936.

"The glaucomatous eye is characterized not only by high intra-ocular tension but also by ready fluctuation of tension. This instability of tension is known to occur very frequently in the otherwise healthy mate of a definitely glaucomatous eye." Ohm (with the ophthalmologic clinic of the University of Berlin) used the following provocative tests:

the drinking test of Marx, the caffeine test and the dark-light test. The material consisted of eighteen cases of primary glaucoma, seven of secondary glaucoma, six of pseudoglaucoma, two of instability of tension without other signs of glaucoma and thirteen control cases. The diurnal variations of tension were recorded before making the tests. During the course of the drinking test and the caffeine test, but not of the dark-light test, even the controls showed elevation of tension up to 30. Primary glaucoma always reacted more strongly than secondary glaucoma. The reaction to the dark-light test was less pronounced than that to the other two tests. It did not seem to depend on the depth of the anterior chamber.

As instances of intermittent glaucoma the author describes cases in which the response to provocative tests varied from one time to another. The eyes were definitely glaucomatous. The reviewer doubts the appropriateness of the term "intermittent" for glaucoma of this description.

As examples of pseudoglaucoma were classified six cases with glaucomatous excavation, atrophy of the optic nerve and defects of the visual fields but with normal tension and normal response to the provocative tests. In three of these cases deposits of lime were visible roentgenologically in the region of the intracranial portion of the internal carotid artery. In one of these cases high intra-ocular tension was shown on several occasions.

As a practical procedure to diagnose early glaucoma, he recommends, first, study of the diurnal fluctuations; if these are slight, the drinking and the caffeine test are indicated. Since the response of normal eyes to these tests is only quantitatively different from that of glaucomatous eyes the disturbance which underlies glaucoma must be an ocular one.

P. C. KRONFELD.

Hygiene, Sociology, Education and History

DEFUNCT LONDON EYE HOSPITAL. A. SORSBY, *Brit. J. Ophth.* 20:77 (Feb.) 1936.

This lengthy historical article does not lend itself to abstracting. The reader, however, will find himself repaid by reading it.

W. ZENTMAYER.

THE CINEMATOGRAF AND THE VISUAL APPARATUS. J. A. GALLINO, *Arch. de oftal. de Buenos Aires* 10: 365 (June) 1935.

This detailed official report based on the statistics of the Educative Cinema Institute of Italy and on the opinions of several Italian, French and Dutch ophthalmologists specifies the apparatus to be employed and the classroom conditions requisite for the proper use of the cinematograph in school teaching.

Gallino reaches the following conclusions: 1. The cinematograph is not injurious to the eyes of school children if their eyes are sound. 2. Its employment should be barred for children with myopia, astigmatism of high degrees, disturbances of accommodation and of binocular

vision, muscular insufficiencies and active ocular disease. 3. There is no objection to its employment for children whose refractive defects are corrected. 4. The duration of each part should not exceed a period of from ten to fifteen minutes, the interval between the parts being from two to three minutes. 5. The screen should be located at a distance of 5 meters from the children. 6. The illumination of the room should be so arranged that the transitions are gradual, and a slight general illumination is convenient during the projection of the film. 7. The condition of the films and of the apparatus should be periodically inspected by experts. 8. The total duration of the filming should be limited to one hour. 9. The apparatus should be simple, of low cost and durable.

C. E. FINLAY.

Injuries

OCULAR LESIONS DUE TO HIGH FREQUENCY CURRENTS. O. BARRATTA, *Ann. di ottal. e clin. ocul.* 64:299 (May) 1936.

Barratta observed three cases of ocular lesions due to high frequency currents. In all three cases there were localized burns about the eye as a result of the current. In the first case the vision began to diminish immediately after the accident, and after four months the right eye showed a complete cataract, which had swollen so as to abolish the anterior chamber and cause secondary glaucoma. The left crystalline lens at this time showed some punctate opacities. The lens of the right eye was removed following an Elliot trephining, resulting in vision of 1/10. In the left eye the opacities progressed slowly, not requiring operation until three years later, when a good result was obtained.

The second case was observed three months after the accident, when vision was reduced to 1/10 in each eye. The lens opacities affected the anterior cortex and posterior cortex just under the capsule, the nucleus being clear. Nine months after the accident the lenses were almost completely opaque.

The third case was interesting, as only a localized opacity in the lens was present after fourteen months. This affected the anterior cortex in a sector running out from the center of the lens. There were also a few subcapsular vacuoles on the nasal side. Vision was reduced to 6/10. Two months later the vision had improved to 10/10, although the appearance of the lens opacities was about the same. This patient also showed miosis on the side of the lens opacities, which by pharmacologic tests the author proved was due to paralysis of the sympathetic nerve endings in the iris.

A bibliography accompanies the article.

S. R. GIFFORD.

MAGNETIC INTRA-OCULAR FOREIGN BODIES AND EXTRACTION WITH THE ELECTROMAGNET. J. SELLAS, *Arch. de oftal. hispano-am.* 36: 17 (Jan.) 1936.

Sellas' statistics of incidence from 1925 to 1934 yielded only 2.5 per thousand; he considers the occurrence rare. The diagnosis is generally easy; exceptionally, however, the insignificance of the injury, its situation, the presence of other lesions or processes, the existence of

inflammatory exudates and a defective history cause the foreign bodies to be overlooked. Whenever there is the slightest doubt roentgen examination is indispensable. For the localization of such bodies Sellas employs a simple method involving subconjunctival introduction of leaden threads. The prognosis is always serious, Sellas' statistics indicating loss of sight in 44 per cent of the cases, and those compiled from several other authors, in 43.5 per cent. As to treatment, he considers as early as possible extraction of the foreign bodies most urgent. When they are localized in the anterior segment of the eyeball they should be removed through the original wound or, if this is closed, through a keratome incision; those in the posterior segment he is inclined to extract through a scleral incision. Except in cases in which the situation is very superficial, in which a hand magnet can be used, he employs a giant magnet, his own model, constructed on the lines of Haab's. In the treatment of the coexisting inflammatory conditions he uses protein therapy, subconjunctival and intravenous injections of mercuric cyanide, autohemotherapy and the usual local medication. The resulting lesions, such as traumatic cataract, he treats on the usual lines.

Appended to the paper is a detailed statistical report of fifty-nine cases. In 44 per cent of the cases there was loss of vision, in 23.72 per cent recovery with normal vision and in 32.88 per cent recovery with reduced vision.

C. E. FINLAY.

A RARE LESION OF THE EYE AFTER TREATMENT WITH ROENTGEN RAYS. A. PILLAT, *Klin. Monatsbl. f. Augenh.* 94: 384 (March) 1935.

A woman aged 51 was treated with roentgen rays for a retrobulbar tumor. Crossed rays were applied from three fields of entrance, each field receiving 7,000 superficial roentgens. Two years later a typical posterior rosette cataract developed, which was followed by general opacification of the lens. After extraction of the cataract a change resembling disciform degeneration was found in the macula; it was surrounded by a circle of small retinitic foci. The fundus of the second eye was free from pathologic change.

K. L. STOLL.

Instruments

A PORTABLE SCOTOMETER, J. P. S. WALKER, *Brit. J. Ophth.* 20: 466 (Aug.) 1936.

The apparatus folds into a case in which there is room for spare charts and the rod carrying the "moving spots." There is also a chin rest. The chart holder has a small hole in the center, behind which there is a small electric bulb. The chart also contains a center hole. The chart is inserted with a matt black surface toward the patient. The moving spot of light is obtained by means of a rod on the tip of which is a small hole. Behind this is a rotating disk with colored glasses, transilluminated by a pocket battery in the handle. Behind the hole is a pinpoint with a spring guard, and below it is a square block of metal. The rod is placed over the chart, keeping the block in contact

with the chart. As soon as the patient says that the light has gone, a slight pressure against the chart with the rod will bring the pinpoint into action and a prick will be made on the chart.

The article is illustrated.

W. ZENTMAYER.

Lens

"POSTERIOR NEEDLING" IN THE TREATMENT OF LAMELLAR AND OTHER FORMS OF SOFT CATARACT. A. J. BALLANTYNE, *Brit. J. Ophth.* 20: 540 (Sept.) 1936.

Ballantyne states that the operation described has been practiced in Glasgow for many years. The usual preparation of the eye is made, and the pupil is dilated with atropine. Cocaine and epinephrine are used to obtain anesthesia. The room should be darkened and focal illumination secured by means of a hand lamp. Any sharp knife needle may be used. The needle is passed through the sclera below the tendon of the external rectus muscle and 5 mm. from the corneoscleral junction, its point being directed into the vitreous forward to the posterior pole of the lens. Then, by carrying the handle of the needle backward in the direction of the temple, the point is turned forward and can be seen to enter the substance of the lens. A criss-cross incision is made in the posterior capsule and cortex of the lens, and the needle is withdrawn in the reverse of the direction of entry. Naturally the operation occupies only a fraction of the time required to describe it. The eye is usually bandaged for the first twenty-four hours, after which the bandage can be removed. Atropine is continuously applied.

While there may be some uncertainty as to the result of a single needling, and it usually has to be repeated, one can be certain that the patient will be spared the suffering incident to the occurrence of iritis and secondary glaucoma. The patient may be treated as an outpatient.

To the author's knowledge, in only one case was a detachment of the retina discovered, and this was some years after the operation.

W. ZENTMAYER.

ASTIGMATISM OF THE LENS. Y. SHOJI, *Ann. d'ocul.* 173: 467, 1936.

The crystalline lens is not a perfect unit in an optical sense, as the index of refraction varies. The pupil is much smaller than the diameter of the crystalline lens, and the light rays that traverse the periphery of the lens are intercepted by the iris screen.

Astigmatism of the cornea is daily met with, and one understands without difficulty that all astigmatism is due to the cornea. However, in the presence of a cataract one often notes an irregular astigmatism due to modifications of the lens. The encyclopedia notes astigmatism of the lens briefly, but in practice not much attention is paid to it.

In a period of five years Shoji met with three cases of regular crystalline astigmatism, and he recently observed another case discovered by one of his colleagues.

The degree and axis of the crystalline astigmatism does not always accord with those of the corneal astigmatism. The glasses preferred by

patients do not always accord with those determined by optical examination. The visual clearness obtained with corrective glasses does not always reach normal.

The details of four cases are given in tabular form.

S. H. McKEE.

DEMONSTRATION OF LATENT DYSTROPHY OF THE CRYSTALLINE LENS IN AVITAMINOSIS. C. G. MOURIQUAND and J. ROLLET, *Compt. rend. Soc. de biol.* 122:1118, 1936.

In scorbutic guinea-pigs corneal paracentesis induces within twenty-four hours minute foci of cloudiness in the lens, visible with the slit lamp. If the animals are then given vitamin C, the mottling disappears completely, but if the administration of the vitamin is delayed three days, the lesions tend to persist.

J. E. LEBENSOHN.

PRINCIPLES INVOLVED IN THE EXTRACTION OF CATARACTS. E. SEIDEL, *Arch. f. Ophth.* 135:159 (Jan.) 1936.

Seidel believes that postoperative iridocyclitis is not caused by lens matter left in the eye but by infection, and that the shock of the operation can be lessened by more complete anesthesia and by shortening and simplifying the surgical procedure. His procedure is as follows: maximum dilation of the pupil with atropine; repeated instillation of 10 per cent cocaine; fixation of the globe just below the counterpuncture; large (over two-fifths) limbic incision with formation of a conjunctival flap continuous with the cornea; removal of a big piece of lens capsule; expression of the nucleus and of what cortex comes out with it; no scooping out of the remnants and no iridectomy; instillation of 1 per cent physostigmine. The author has had only 2 prolapses of the iris in 140 extractions. He stresses the dangers of the intracapsular method.

P. C. KRONFELD.

Methods of Examination

KINESCOPY—OBJECTIVE AND SUBJECTIVE (SUPPLEMENTARY REMARKS). S. HOLTH, *Brit. J. Ophth.* 20:412 (July) 1936.

These are brief supplementary remarks to the author's earlier paper (*Brit. J. Ophth.* 19:603 [Nov.] 1935). It is not possible to abstract this short paper satisfactorily. It should be read and compared with the original paper.

W. ZENTMAYER.

THE FIRST RADIUS-FIGURE FOR SUBJECTIVE ASTIGMOMETRY. S. HOLTH, *Brit. J. Ophth.* 20:415 (July) 1936.

This is a brief historical memorandum calling attention to the radius figure for estimating astigmatism at near distance first published by Javal in 1865. The examination was made binocularly with two objects, namely, a circle before one eye and radiating lines before the other; the intention of the procedure was to suspend convergence and accommodation, making atropine superfluous. It is this radius figure, magni-

fied for the distance of 6 meters and surrounded by the clock periphery (radiating lines for every hour and for every half hour), which even today is used by nearly all oculists for subjective astigmatometry.

W. ZENTMAYER.

A PHOTO-ELECTRIC METHOD TO STUDY THE OXYGEN CONTENT OF THE BLOOD IN THE EYE. G. SCHUBERT, *Arch. f. Ophth.* 135: 558 (July) 1936.

A photo-electric cell sensitive to red rays is put opposite the dilated pupil of a rabbit's eye, which is illuminated discially by means of the Lange lamp. The photo-electric cell is connected with a sensitive galvanometer, which registers a current that depends on the amount of light emitted by the rabbit's fundus. Shutting off the oxygen to the rabbit lowers the intensity of this current, while slowing the retinal circulation increases the current. Schubert recommends this method for determining the degree of the oxygen saturation of the blood in the eye.

P. C. KRONFELD.

Neurology

CILIARY (MIGRAINOUS) NEURALGIA AND ITS TREATMENT. WILFRED HARRIS, *Brit. M. J.* 1: 457 (March 7) 1936.

Ciliary neuralgia is a form of migrainous neuralgia in which the pain is located in or about the eyeball. In addition, the eye may become extremely congested and reddened and show lacrimation. The neuralgia is usually recurrent. It affects the temple and side of the forehead and extends to the back of the head. The pain is intense and excruciating. It may last for from ten minutes to thirty hours. Nausea occasionally accompanies the pain, but visual spectra and transient hemianopia are absent. The condition can be helped and attacks arrested for months or years by injecting alcohol into the supra-orbital or intra-orbital nerves; an injection into the gasserian ganglion, which produces permanent trigeminal anesthesia, is more apt to give a lasting result. This condition should be distinguished from trigeminal tic involving the ophthalmic division of the fifth cranial nerve and chronic neuralgia of the jaw. Other painful conditions in the region of the eye which must be distinguished are glaucoma, herpes frontalis and leaking aneurysm of the circle of Willis (Bramwell, Edwin: *Tr. Ophth. Soc. U. Kingdom* 54: 205-221, 1934).

Harris believes that migrainous neuralgia is due to vasomotor influences on the dural blood vessels, especially on the middle meningeal artery.

He has observed twenty-three cases of ciliary neuralgia, which occurred practically in all periods of life, perhaps most frequently in that between 40 and 50 years. The onset of pain is usually in the early hours after midnight. In ten of these cases the painful eye was red and intensely congested, often showing lacrimation.

Harris concludes with the following summary: "Migrainous neuralgia is a substituted form of migraine, in which the pain is referred

anteriorly into the temple, eye, cheek and jaw. It is usually strictly unilateral, though occasionally it may pass over the opposite side at the end of an attack, as in migraine, another point of resemblance being the not uncommon presence of nausea. There are never present any of the cerebral phenomena of teichopsia, hemianopia, aphasia, or monoplegia. This difference in its symptoms may be due to vasomotor spasm affecting blood vessels of the dura, such as the middle meningeal instead of the posterior cerebral artery as in migraine, the pain in migrainous neuralgia being referred along the recurrent trigeminal meningeal branches. Such a view of its pathology would account for the good effect undoubtedly produced in many cases of severe migrainous neuralgia by alcohol injection of the supra-orbital or infra-orbital nerves, or by injection of the inner two-thirds of the Gasserian ganglion, which produces a more lasting cure. In many cases the duration of the paroxysms may be as short as ten minutes or a quarter of an hour, sometimes from four to six attacks occurring in the twenty-four hours. It is such brief attacks that might possibly lead to confusion of the diagnosis with trigeminal tic. In certain cases of migrainous neuralgia the pain is especially referred into the eyeball and around the eye, and in a large proportion, nearly 50 per cent, the eye becomes very congested with lacrimation—a veritable ocular crisis.”

A. KNAPP.

Operations

CORRECTION OF PTOSIS BY TWO STRIPS OF FASCIA LATA. J. A. MAGNUS, *Brit. J. Ophth.* 20:460 (Aug.) 1936.

Magnus describes the operation introduced by Lexer of Munich in 1923 (*Klin. Monatsbl. f. Augenh.* 70:464, 1923). The advantage of Lexer's technic is that a direct connection is made between the frontalis muscle and the lid margin by a tissue which can be left in place. In time the tissue undergoes a natural contraction which emphasizes the effect of the operation. For practical purposes the article should be consulted, as it is illustrated.

W. ZENTMAYER.

KERATOPLASTIC REPARATION OF THE ENTIRE CORNEA. R. FRIEDE, *Arch. f. Augenh.* 109:662, 1936.

Friede reports six cases in which he transplanted a large piece of cornea from one human being to another, with results which satisfied him that the procedure was feasible. Some of the transplanted pieces of cornea were taken from postmortem material. Every patient treated had a total leukoma, and from 9 to 11 mm. of the opaque cornea was excised with a trephine and replaced with a transplant of similar size, similarly excised. The technical details are given in full, with a general discussion of corneal transplantation. The protocols do not announce any improvement in visual acuity, but Friede believes that this procedure may save eyes that have perception of light with total leukoma from eventual enucleation. In most of the cases previous keratoplastic operations had been performed without success.

F. H. ADLER.

Orbit, Eyeball and Accessory Sinuses

THE CAUSE OF VOLUNTARY FORWARD LUXATION OF THE EYEBALL.
D. J. LYLE and J. S. McGAVIC, *Am. J. Ophth.* 19: 316 (April) 1936.

The authors review the literature and report the case of a colored man who could voluntarily luxate the right eye. He died of pneumonia, and an autopsy was secured. The left orbit was found to be normal. In the right the superior oblique muscle was found to bifurcate, the lower branch going to its normal insertion while the upper was attached to the superior rectus muscle 10 mm. behind its normal insertion. In the lower part of the orbit was a protractor muscle, which arose just behind the orbital tubercle and was inserted in the posterior part of the globe.

W. S. REESE.

GRANULOMA OF THE BULBAR SUBCONJUNCTIVAL TISSUE ARISING FROM AN EMBEDDED CILIUM. F. W. G. SMITH, *Brit. J. Ophth.* 20: 455 (Aug.) 1936.

The patient was a woman aged 66 years. There was no history of injury, and the condition had been diagnosed as episcleritis. There was a localized swelling, solid and slightly gelatinous in part. It was beneath the conjunctiva. A number of large vessels radiated from the growth. With the loupe a body of different refractive index was seen in the gelatinous portion and proved to be a cilium, one of her own. The article is illustrated.

W. ZENTMAYER.

Pharmacology

SYNTHETIC SUPRARENIN BITARTRATE AS A MYDRIATIC. W. D. HORNER and J. BETTMAN, *Am. J. Ophth.* 19: 311 (April) 1936.

The literature on epinephrine and substitutes for it is reviewed especially as to the clinical uses and physiologic effects. Following the use of suprarenin bitartrate on seventy-four patients in a home for the aged the following conclusions were reached:

1. For ophthalmoscopic work, 2 drops of 2 per cent bitartrate are sufficient and will not only dilate the pupil as much and as rapidly as a larger dose but may be easily controlled by physostigmine. This dose would seem ample for mydriasis except when adhesions of the iris must be broken up.

2. The amount of the drug has little influence on the speed of dilatation.

3. Mydriasis from 2 drops lasts half again as long (twelve and three-quarter hours) as that from 1 drop (eight and a half hours); 4 drops give the same result as 2 drops.

4. A pupil dilated with 2 drops of this drug will return to normal in about twenty-five minutes if 1 drop of physostigmine is instilled twice at ten minute intervals.

5. Headaches are common after administration of suprarenin bitartrate (in 54 per cent of the cases in the authors' series) and are more severe when physostigmine is used in combination with it.

6. A combination of suprarenin bitartrate and powdered atropine is recommended when a supermydriatic is desired.

W. S. REESE.

INHIBITION OF EPINEPHRINE AUTO-OXIDATION BY AQUEOUS. F. BONHOMME, *Compt. rend. Soc. de biol.* 122: 110, 1936.

Epinephrine hydrochloride (1:100,000) buffered at p_H 6.77 oxidizes rapidly in air; in a few minutes some coloration appears; in one hour, definite redness. After nineteen hours the preparation is totally inactive, whereas the same concentration of epinephrine in aqueous similarly buffered and exposed has conserved most of its biologic potency. Blood plasma has an even greater protective action than aqueous. The preservative properties seem largely due to the proteins present, not to vitamin C or other reducing agents.

J. E. LEBENSOHN.

The Pupil

THE AMAUROTICALLY FIXED PUPIL: I. ITS USE AS A PHARMACOLOGIC TEST OBJECT. F. POOS and H. GROOSE-SCHÖNEPAUCK, *Arch. f. Ophth.* 135: 144 (Jan.) 1936.

In studying the effect of drugs and of endogenous factors such as the carbon dioxide tension or the epinephrine content of the blood, it is always difficult to keep the other factors which influence the width of the pupil unchanged. Besides, the double innervation of the sphincter introduces a certain factor of ambiguity in that, for instance, dilation of the pupil may be the result either of a lowered parasympathetic or of an increased sympathetic tone. In other words, for experimental purposes simplification of the normal pupillary mechanism is desirable. The simplification introduced by the authors of the paper under review is unilateral amaurotic fixation, produced by transclerally destroying the retina around the disk with a small curet. This amaurotically fixed pupil is a most satisfactory pharmacologic test object, on which the effect of epinephrine, cocaine, atropine, pilocarpine and physostigmine have been studied.

P. C. KRONFELD.

Physiology

RELATIONSHIP BETWEEN THE PRESSURE IN THE VEINS ON THE NERVE HEAD AND THE PRESSURE OF CEREBROSPINAL FLUID. F. GIBBS, *Arch. Neurol. & Psychiat.* 35: 292 (Feb.) 1936.

Gibbs confirms Baurmann's work by showing a direct relationship between the pressure of the cerebrospinal fluid and the pressure in the retinal veins at the optic disk. Experiments were conducted on four cats and four dogs. A hollow needle connected to a reservoir of physiologic solution of sodium chloride was inserted into the anterior chamber. By raising the level in the reservoir the intra-ocular pressure was raised until a vein on the optic nerve showed evidence of collapse.

The pressure necessary to cause partial collapse was taken as the venous pressure. The pressure of the cerebrospinal fluid was varied by means of a pressure bottle connected through a hollow needle with the cisterna magna. Alterations of cerebrospinal fluid pressure below 18 mm. of mercury did not affect the pressure in the central retinal veins, but when the pressure of the cerebrospinal fluid was raised above 18 mm. the intra-ocular venous pressure rose step by step with it, remaining from 2 to 4 mm. above it, until the pressure of the cerebrospinal fluid was greater than the intra-ocular arterial pressure.

Gibbs suggests the application of this relationship for indirect determination of high cerebrospinal fluid pressure when it seems undesirable to enter the cisterna magna or the lumbar sac. He stresses that the relationship is of theoretical interest because it shows that the pressure in veins with truly intracranial drainage varies directly, within broad limits, with the pressure of the cerebrospinal fluid.

R. IRVINE.

THE CENTER OF ROTATION OF THE EYE. M. C. COLENBRANDER, Arch. f. Augenh. 109: 622, 1936.

Colenbrander reviews the previous literature on this subject and reports his experiments to determine the center of rotation in his own eye. The visual lines plotted by his subjective method cross in the eye in an area which is 1.3 mm. high and 0.9 mm. broad. No retraction of the globe was found during the movements, and the center of rotation was proved to be not eccentric. He concludes that for all practical purposes one can assume that with the head held upright there is a simple point or center of rotation.

F. H. ADLER.

A SCHEMA FOR EYE MOVEMENTS. M. C. COLENBRANDER, Arch. f. Augenh. 109: 629, 1936.

Colenbrander assumes that the movement of the eye in any direction from any chosen position can be expressed as a rotation around three independent axes. In order to analyze the effect of each muscle in this movement, one needs to know only the position of the muscle in respect to these axes. Usually one thinks of the eye as turning in the orbit, which is stationary, but the calculations remain the same if one imagines the eye remaining stationary and the orbit turning around it. The origin of each muscle then changes its position in space, and one can then calculate this position according to Listing's law.

Colenbrander used Volkmann's figures for the origin and insertion of the ocular muscles and from his calculations arrives at the following conclusions: The two oblique muscles rotate the eye (torsion) in every position, but the torsional effect is much less when the eye is in the adducted position. The internal and external rectus muscles have a pure adducting and abducting effect only when the eyeball is in the horizontal position. When the eye is in any other position they cause either elevation or depression of the globe and also have considerable torsional effect. The superior and inferior rectus muscles cause adduction when the globe is turned in and abduction when the globe is turned out. The borderline where adduction changes to abduction depends on the degree to which the eye is elevated or depressed.

F. H. ADLER.

Refraction and Accommodation

THE RELATION OF ACCOMMODATION TO THE SUPPRESSION OF VISION IN ONE EYE. G. A. FRY, *Am. J. Ophth.* 19: 135 (Feb.) 1936.

This is a continuation of the work undertaken by McDougall in 1903. Fry gives the following summary and conclusions:

"The voluntary suppression or favoring of vision in one eye has been demonstrated to be mediated through changes in accommodation by showing that the voluntary control is abolished by paralyzing the ciliary muscles with homatropine. In the case of afterimages the control is brought about through changes in intraocular pressure because optical blurredness is not involved, and because it can be shown that applying pressure to one of the eyes causes the impression of that eye to predominate over that of the other eye. In the case of the rivalry of impressions produced by direct stimulation, the voluntary control is mediated through changes in the blurredness of the optical image, because voluntary control is abolished by use of a small artificial pupil, which minimizes the effects of changes in accommodation upon optical blurredness but does not interfere with proprioceptive impulses of changes in intraocular pressure."

W. S. REESE.

Retina and Optic Nerve

JENSEN'S CHORIORETINITIS. G. BOSSALINO, *Arch. di ottal.* 43: 1 (Jan.) 1936.

The literature on choroiditis juxtapapillaris is reviewed, and four cases are reported. These instances occurred in young women aged from 15 to 22. In all, the characteristic fan-shaped scotoma was present, extending to the periphery and indicating a lesion involving the whole thickness of the retina, including the nerve fibers. In all, the single lesion was near the disk, the disk itself showing edema. In the first case the area of retinal edema disappeared completely in three months, leaving no visible scar, but the scotoma persisted. In the others slight pigmentary changes occurred. In two cases signs of iritis were also present with some precipitates. All active symptoms subsided in all the patients within five months, the minimal scars indicating that the lesion is primarily in the retina with very little disturbance of the pigment epithelium.

An interesting comparison is made between these cases and a case of choroiditis in which a very large lesion produced only a central scotoma, leaving the peripheral field normal.

In two of the patients with typical changes tuberculin tests were positive, and in the other the condition was considered as possibly due to tuberculosis, which is generally considered to be the commonest cause of the condition.

S. R. GIFFORD.

LEBER'S HEREDITARY ATROPHY OF THE OPTIC NERVE. HORACIO B. MOULIÉ and JUAN HURTAULT, *Arch. de oftal. de Buenos Aires* 10: 677 (Sept.-Oct.) 1935.

The authors refer to the symptoms and to the usual matriarchal heredity in male members, with only occasional occurrence in the females,

as pointed out by Bell, and review the literature. They then present four cases of atrophy of the optic nerve in three males and one female in one family (another brother and sister had died in infancy), with the atrophy coming on at from 8 to 15 years of age, after an acute onset, and coming to a standstill with permanent reduction of vision and a central scotoma. Neither their parents nor their children showed any signs of the disease; the latter were, however, all under 14 years of age. The serologic and clinical data were irrelative.

C. E. FINLAY.

LAGLEYZE-VON HIPPEL'S DISEASE AND PIGMENTARY RETINITIS.
ESTEBAN ADROGUÉ and JORGE MALBRÁN, Arch. de oftal. de Buenos Aires 10: 692 (Sept.-Oct.) 1935.

This is a review of the different manifestations of retinal angiomatosis. Priority in the description of this disease the authors, with other Argentine writers, claim for Lagleyze, who described it in 1884, von Hippel not describing the disease till 1903. They reproduce Lagleyze's description of his case, in connection with which they find that the family history indicates pigmentary telangiectasia of the skin and epistaxis in a sister, one of her sons and her grandson and retinitis pigmentosa in another of her sons and in a daughter, nine other children showing no ocular lesions. Seven brothers of Lagleyze's patient and the parents of the patient showed normal eyes.

The authors review the different classifications of telangiectatic tumors made by different authorities and the published reports of cases of retinal angiomatosis in some of which there were similar lesions elsewhere. They consider the ophthalmoscopic picture described by Lagleyze and von Hippel as the most common one. The lesion may also show as numerous miliary aneurysms, as depicted by Oeller, as aneurysmic formations in the vicinity of the disk and as racemose aneurysm of the retinal vessels. They incline to consider the vascular lesion as primitive and the glial one as secondary and are supported therein by the relative benignity of similar tumors in the brain. The different points of view from a pathogenic standpoint are also reviewed.

C. E. FINLAY.

CYSTOID DEGENERATION AND CYSTS OF THE RETINA. J. CASANOVAS,
Arch. de oftal. hispano-am. 36: 239 (May) 1936.

Microscopic examination of eyes frequently reveals cystic degenerations of the retina. These are brought about by different pathologic processes but have certain features in common. In many instances the lesion is not actually cystic but edematous. Retinal cysts are considered as a terminal transformation of cystic degenerations. These are studied from an anatomicoclinical point of view.

The most frequent form of cystic degeneration is that observed in the vicinity of the ora serrata, diminishing centrally, in persons of advanced age. As age progresses the affected areas are apt to coalesce and form a ring, the width of which progresses with advancing years. The situation is too peripheric for ophthalmoscopic examination unless the retina is detached and then only with red-free light (Vogt).

Although more frequent in old persons, the cysts are also found with relative frequency in persons of 30 and even less. The earliest lesion consists in a rarefaction of the nervous elements, giving the retina a reticulated aspect; next small cavities are formed, which gradually increase in size, coalesce and invade the different retinal layers. The cystic contents consist of a serous, fibrinous and at times gelatinous fluid. Perforation of the membranae limitantes never occurs. There are great individual variations. The variations reported by different authors are reviewed in detail.

Casanovas refers to the different theories explanatory of the pathogenesis, the prevalent opinions being divisible into those according to which the primary process is atrophic and those according to which the lesion is attributable to lymphatic stasis. To the latter he inclines.

The macular region follows the retinal periphery as the most frequent site for the occurrence of cystic degeneration, its anatomic structure favoring a separation of its layers (E. Fuchs). The initial period is represented by a honeycomb aspect of the macula, observable only with red-free light, mostly in the yellow area and less frequently in the foveal or the perifoveal zones. A development of this lesion leads to cystic degeneration of the macular region. When the condition is complicated with hemorrhages extravasated blood is found in the cavities. A still further development leads to swelling of this region with a characteristic aspect and change in the direction of the skiascopic reflex (Adrogué and Sená). Large cysts can be mistaken for holes in the retina, but the differential diagnosis can be made with red-free light, which permits observation of the cyst wall.

Casanovas next refers to holes in the macula and to their formation from cysts, as maintained by different authorities.

Cystic degeneration of the macula may appear as an isolated lesion or as a development complicating other retinal pathologic processes such as retinitis pigmentosa or retinitis punctata albescens.

An anatomic examination of the lesion was carried out by Casanovas in three eyes enucleated for corneal ulcer, iridocyclitis and septic endophthalmitis in which it was localized in the external plexiform layer and produced by excessive transudation from the capillaries. E. Fuchs has also found it in glaucoma. Nuel's vesicular edema of the macula is also a cystic degeneration of the retina. Likewise in Kuhnt's central atrophic retinitis the hole formation was undoubtedly prepared by cystic degeneration.

The lack of blood vessels at the macula and at the retinal periphery, as well as their extreme thinness, explains the special vulnerability of these regions.

Cystic degeneration can also be found in other regions of the retina, with more or less diffuse distribution, having been reported in retinitis circinata, in albuminuric, diabetic and cachectic retinitis, in Coats' disease, in the Hippel, Lindau and Recklinghausen retinal complications, in tuberous cerebral sclerosis with localizations in the retina, optic nerve, chiasm and brain, and in choroidal sarcoma. In some of these conditions it may be the cause of a tear with subsequent detachment of the retina. On the other hand, in retinal detachment the substitution

of nerve tissue by glial tissue favors cystic degeneration. Casanovas reports anatomic examination of the retina in several illustrative cases.

Fully developed cysts of the retina can be congenital or acquired. The former are usually associated with malformations such as coloboma or microphthalmos. The acquired can be of parasitic origin (*cysticercus*) or connected with cystic degeneration. Several cases reported by different authors in connection with detachment of the retina are referred to. Most authors consider the cysts as a macroscopic development from cystic degeneration with destruction of the separating septums. Casanovas gives several detailed anatomic descriptions. C. E. FINLAY.

PRESENT STATUS OF THE TREATMENT OF RETINAL DETACHMENT. H. ARRUGA, Arch. de oftal. hispano-am. 35: 514 (Oct.) 1935.

Arruga reviews the present status of the operative treatment of retinal detachment, which is based on the closure of the retinal tears and now yields recoveries in over 50 per cent of the cases. He insists on the necessity of a prior careful clinical study of the patient from a general and a local point of view. The percentage of cures in selected cases reaches 70, the most favorable conditions being: satisfactory general and local conditions, a traumatic origin, absence of any local reaction, and short duration. He reviews the general and local contraindications, which he tries to correct when possible without losing too much time.

He describes the preparation of the patient, the instruments, the method of anesthetization and the operative procedure. In the latter, according to the individual indications, he uses thermocautery or galvanocautery, the Vogt points, diathermy (Weve, Šafář or Lacarrère's electrodiaphase) or scleral trephining. In connection with the post-operative precautions he stresses the importance of local quiet. He reviews the possible complications.

He refers to his statistics for the period from 1932 to 1934, during which he operated on 248 patients with 168 replacements of the retina, obtaining in 141 vision over 0.15.

He concludes that by operative treatment recovery is obtained in 50 per cent of the cases, success depending on prompt execution, correct localization of the tears and their isolation by operative procedure. He considers the method employed of secondary importance but considers that diathermy offers the best chance for recovery.

C. E. FINLAY.

CURE OF DETACHMENT OF THE RETINA CAUSED BY A HOLE IN THE MACULA BY CATHOLYSIS: REPORT OF CASES. A. VOGT, Klin. Monatsbl. f. Augenh. 96: 15 (Jan.) 1936.

Vogt discusses the advantages of his cathodal electrolysis, which he introduced in the operative therapy of detachment of the retina two years ago. One of these advantages is that bubbles of white foam aid in ophthalmoscopic localization and orientation. The value of this method in the closure of retinal tears has been proved. It is especially valuable in those cases in which a hole in the macula is the cause of a detachment of the retina. In the treatment of macular tears it is superior to chemical methods, in which the visual results are unsatis-

factory owing to the destruction of the retina. For the same reason and on account of the loss of vitreous following it, Vogt considers diathermy less reliable in these cases. He reports the case of a woman aged 26 with binocular myopia of 28 diopters and with a detachment of the retina of a spontaneous or perhaps traumatic nature. A hole in the macula the size of the diameter of a large retinal vein was closed by means of catholysis and an angular needle in two sittings, which took place seventeen days apart. The scars in the macula appeared as two faint dots, which did not interfere with the vision, the latter amounting to from $4/25$ to $4/30$ of the normal after correction with a concave lens of 28 diopters. This method was employed in another case in which there was a hole in the macula caused by cystoid degeneration in a woman aged 62. The vision was $1/10$ of the normal before the operation and from $5/20$ to $5/12$ after closure of the hole, which had a diameter of about one half of a disk diameter.

K. L. STOLL.

BILATERAL DETACHMENT OF THE RETINA WITH SYMMETRICAL TEARS OF THE ORA SERRATA IN TWO NONMYOPIC YOUTHFUL BROTHERS: THE DOUBTFUL RELATION TO ISOLATED CYSTS OF THE RETINA. H. SCHMELZER, *Klin. Monatsbl. f. Augenh.* 96: 19 (Jan.) 1936.

Two brothers aged 26 and 33 had suffered from defective vision, the younger for a few months prior to admission to the hospital, the elder for several years. These brothers resembled one another as to color of the eyes and hair, shape of the face, height and other traits common usually in "two-egg twins." A twin brother of the younger man, however, differed totally from him in appearance and had healthy eyes. The left eye of the younger patient was slightly hyperopic; the other eye and both eyes of the older patient were emmetropic. In each patient the bilateral detachment of the retina was shallow, was located in the lower portion of the eyeballs and was well defined upward. In each patient the bilateral tear at the ora serrata occurred in the temporal and lower periphery of each eye; the tears in the two patients were identical even in that the tear in one eye in each patient was subdivided by some kind of remaining trabeculae of the retina. This can be observed in the photogram of the right fundus of the younger brother and in that of the left of the elder. This appearance prompted Schmeltzer to consider whether these tears might have developed on primary solitary retinal cysts such as were described recently by Weve. Etiologically, in the author's opinion, symmetrical tears of the retina are based on a developmental disturbance—probably on an abnormal "anlage" of the retina. This view appears to be supported by the anamnesis: the resemblance of these two brothers, their emmetropia and the absence of any ocular or bodily trauma. A lesion intra partum could be ruled out, as the patients were delivered without forceps. K. L. STOLL.

IMPORTANCE OF THE CATHODE AND ANODE IN THE ELECTROLYTIC TREATMENT OF DETACHMENT OF THE RETINA. A. VON SZILY and H. MACHEMER, *Klin. Monatsbl. f. Augenh.* 96: 36 (Jan.) 1936.

Von Szily and Machemer discuss the results obtained with the positive and the negative pole, respectively, by Sabbadini, Imre, Vogt and

other authors in electrolytic treatment of detachment of the retina. The results of their own research on rabbits' eyes are described and illustrated with drawings and photomicrographs. No difference of importance was observed between the degrees of inflammatory action obtained with the anode and the cathode. The only practical difference rested in the type and volume of the gas produced and in the celerity of the absorption of the gas. Clinical observation showed that the formation of a large quantity of poorly absorbable gases in the vitreous and in the subretinal space may prevent reattachment of the retina. To prevent this, especially in those eyes which cannot be treated with minimal doses of the current, great care has to be taken in introducing the cathode into the vitreous chamber. The anode deserves the preference because it gives rise to less gas and at the same time produces an equal amount of inflammation. The cathodal needle is not always useful in placing punctures because the retinal focus can be observed less readily around the cathode and the bubbles of gas may change their location where liquefied vitreous is present. The cathode, however, may be attached to the perforating needle when the white focus of the anode cannot be observed in consequence of gray retinal reflexes.

K. L. STOLL.

LYSOTHERAPY FOR RETINITIS PIGMENTOSA. N. K. MANYUKOVA, *Sovet. vestnik oftal.* 8: 349, 1936.

Manyukova applied lysotherapy in forty-three patients who suffered from retinitis pigmentosa. The lysates were made of retina, liver, anterior and posterior hypophyseal lobes, corpus luteum and adrenal cortex. In twenty-four patients the Lüttge-Mertz reaction showed dysfunction of the adrenal cortex, of the posterior lobe of the pituitary gland and of the liver. From fifteen to twenty-five subcutaneous injections of the polylysates were given at the rate of one every third day, the dose being from 0.2 to 0.3 Gm. of each lysate. There was neither objective nor subjective improvement in seven patients. Eleven patients noted a decrease of hemeralopia. In twenty-five patients (58 per cent) the visual acuity and the dark adaptation were improved, and the visual fields were enlarged 5, 10 and 15 degrees in all directions. These effects, however, were transitory, lasting for about two or three months; repeated injections were given to the majority of the patients in the last group.

The conclusions are as follows: The improvement in the visual acuity and the dark adaptation and the enlargement of the visual fields in 58 per cent of the forty-three patients treated make polylysates desirable therapeutic agents in the management of retinitis pigmentosa. The effect of the therapy is transitory. The results in atypical forms of retinitis pigmentosa are more stable.

O. SITCHEVSKA.

Trachoma

THE CARBOHYDRATE MATRIX OF THE EPITHELIAL-CELL INCLUSION IN TRACHOMA. C. E. RICE, *Am. J. Ophth.* 19: 1 (Jan.) 1936.

This study represents an attempt to determine microchemically the nature of the von Prowazek-Halberstaedter inclusion body regardless of

whether it is specifically concerned in trachoma or not. The work is based on observations on seventeen cases of active trachoma at the United States Trachoma Hospital, Rolla, Mo. The following conclusions are given:

"1. The v. Prowazek-Halberstaedter inclusion body in the epithelial cells of trachomatous conjunctivae contains a very appreciable amount of carbohydrate which gives a sharp color reaction with iodine. The evidence seems to indicate that this carbohydrate in the trachoma inclusion body is glycogen. This carbohydrate evidently exists in part as a matrix or diffused throughout the inclusion.

"2. The v. Prowazek-Halberstaedter inclusions may, therefore, be demonstrated with a modified Lugol's solution.

"3. With the weak Lugol's solution, the v. Prowazek-Halberstaedter inclusions can be more certainly demonstrated in thick and indifferent smears than with the use of Giemsa stain. For the clinician, this iodine stain should prove a quick and simple method of examining conjunctival scrapings for inclusions.

"4. It can be demonstrated that the v. Prowazek-Halberstaedter inclusions in wet preparations of unfixed cells contain varying numbers of easily visible granules, after removal of the carbohydrate matrix. These granules are in all probability the 'elementary bodies' of von Prowazek and the so-called 'initial bodies' of Lindner."

W. S. REESE.

STUDIES ON THE INFECTIVITY OF TRACHOMA. R. W. HARRISON and L. A. JULIANELLE, *Am. J. Ophth.* 19:118 (Feb.) 1936.

Harrison and Julianelle give the following summary and conclusions:

"1. A study has been made of the bacteria cultivable from trachoma and clinically similar diseases as well as from normal eyes.

"2. The bacteria cultivable from trachoma are not typical of that infection since they are also recoverable in approximately similar frequency for other conditions of the eye.

"3. While less numerous, the same bacteria may be isolated from the eyes of normal individuals.

"4. The bacterial flora of trachoma does not vary with the different clinical stages of the disease nor with the presence of epithelial-cell inclusions.

"5. Inoculation into susceptible monkeys of all the different varieties of cultures isolated, either individually or pooled, does not induce experimental trachoma even though the tissues from which the bacteria are derived are demonstrably infectious.

"6. Filtrates of infectious trachomatous material when inoculated simultaneously with the organisms isolated exhibit no supplementary nor associated activity capable of rendering the organisms specifically infectious.

"7. The observations made in the present study furnish no evidence, therefore, that any of the bacteria cultivable from trachoma induce the experimental disease in monkeys."

W. S. REESE.

THE SO-CALLED FOLLICLES IN THE SECOND STAGE OF TRACHOMA.
R. J. PULVERTAFT, *Rev. internat. du trachome* 13: 19 (Jan.) 1936.

In stage 1 of trachoma the subepithelial tissue is densely infiltrated by lymphocytes and some plasma cells. These are frequently organized in oval units with central endothelial cell areas. Nothing can be expressed from these solid follicles. In stage 2, the subepithelial infiltration gives way to dense fibrous tissue, and the ducts of the meibomian glands, obstructed in the process, produce bleblike excrescences. These are the "follicles" which are expressible; they are analogous to the cystic changes in acne and rhinophyma.

J. E. LEBENSOHN.

DIAGNOSIS OF RUDIMENTARY PANNUS. A. TRUBIN, *Rev. internat. du trachome* 13: 34 (Jan.) 1936.

Trachoma is essentially a keratoconjunctivitis, and pannus in one form or another is invariably present. For the rapid diagnosis of incipient pannus Trubin suggests examination with a plane mirror ophthalmoscope through a lens of from 10 to 15 diopters. The transmitted light reveals black stripes in the early corneal lesion, corresponding to vessels filled with blood. These stripes become progressively more transparent as the lesion cicatrizes and the vessels empty.

J. E. LEBENSOHN.

Tumors

TUMOR OF THE POSTERIOR POLE ENDURING FOR SEVEN YEARS.
P. VEIL, *Bull. Soc. d'opht. de Paris*, April 1936, p. 302.

Veil completes his observations of a patient whose case was reported to the society in June 1929, June 1930 and March 1933. The interest in the case is due to its long duration and to the fact that the original diagnosis of angioma of the choroid has been confirmed by anatomic findings. The patient, first seen at the age of 50, in 1929, stated at that time that he had had herpes of the left eye complicated by keratitis and paralytic mydriasis in 1926. In 1928 some difficulty in the visual acuity of the right eye resulted in discovery of a central scotoma. Later, vision was reduced to 0.3, with a central scotoma for colors. A small pigmented lesion was found in the macular area, while the inferior and internal portion of the disk had a gray, edematous appearance and dilated veins which showed considerable parallactic displacement. Treatment with mercuric cyanide, neoarsphenamine and iodized poppy-seed oil 40 per cent did not help. The progress was very similar to that in von Hippel's patient (*Arch. f. Ophth.* 127: 27, 1931). However, the tumor appeared somewhat like a small round cell sarcoma, as noted in the illustrations. Veil believes that von Hippel's triad of slow evolution, peripapillar extension and macular alterations does not permit the diagnosis of angioma.

L. L. MAYER.

LYMPHOMA OF THE CONJUNCTIVA. F. TERRIEN, *Bull. Soc. d'opht. de Paris*, April 1936, p. 307.

Lymphomatous tumors of the conjunctiva are very rare. Terrien presented a patient at the last meeting whose lesion proved to be a

lymphoma. The tumor was slightly adherent to the subconjunctival tissue and consisted of a mass of round cells, irregularly flattened, with large nuclei and little protoplasm—thus having the appearance of lymphocytes. Few vessels and little conjunctival tissue were found in the interior of the mass. Around the tumor were dilated, sanguineous capillaries. The observations of Koerber and Cosmettatos (*Ann. d'ocul.* 137: 288 [April] 1907) were entirely comparable, with the difference that Terrien's patient was a man of 60 years while their patients were young adults.

L. L. MAYER.

Uvea

FOREIGN BODIES LIKE "GOLD NUGGETS" ON THE IRIS. R. ROSSANO, *Bull. Soc. d'ophth. de Paris*, December 1935, p. 759.

A patient aged 40 complained of poor visual acuity, which he attributed to the fact that he had a war bullet in his left temporal region. The roentgenogram showed two opaque spots, one in the left orbit near the external parotid gland and the other in the frontal sinus of the left side. The visual acuity of the right eye was 0.3; that of the left, 0.2. The fundi were normal. Examination of the left eye with the slit lamp revealed on the iris, in the inferior temporal sector, near the pupillary border, a number of small brilliant points resembling small nuggets of gold. The cornea was scarred only very slightly. The right eye was normal. Rossano questions that this material is cholesterol because of the absence of siderosis and chalcosis. May it not be silicon?

L. L. MAYER.

FEBRIS UVEOPAROTIDEA (HEERFORDT). A. D. NOWKIRISCHKY, *Klin. Monatsbl. f. Augenh.* 95: 620 (Nov.) 1935.

Heerfordt in 1909 described a syndrome of inflammation of the uvea and parotid gland. The course was subchronic, with persistent low fever, swelling of the parotid gland, stubborn uveitis and frequent paresis of cerebrospinal nerves.

Nowkirischky reports a similar condition in a boy aged 17 in whose family tuberculosis was absent. Both eyes became inflamed consecutively, showing stippled keratitis and iritis with posterior synechiae. Nodules resembling proliferating tuberculous nodules developed in the stroma of the iris near the pupillary margin; there were also round exudative nodules in the same location. The vitreous was cloudy. The parotid glands were swollen and the lacrimal glands enlarged. The roentgenogram showed dense shadows at the hilus and veiling of the lungs suggesting tuberculosis, the presence of which, however, remained uncertain. The condition of the eyes persisted during the hospitalization of the patient from April 16 to May 28, 1934.

Repeated treatments at the hospital and observation for seventeen months failed to establish distinctly whether there was pulmonary tuberculosis, although a tuberculous diathesis was evident and miliary tuberculosis was suspected when the roentgenogram of the right lower lobe of the lung showed small dotted shadows for a certain period.

This patient presenting a number of symptoms suggesting the tuberculous etiology of Heerfordt's disease showed that the disease may be associated with persistent grave ocular symptoms. K. L. STOLL.

Vitreous

CLINICAL STUDIES ON THE VITREOUS: I. THE CONTRACTION OF THE VITREOUS. K. LINDNER, *Arch. f. Ophth.* 135: 332 (May) 1936.

In 1930 before the German Ophthalmological Society Lindner first expressed a theory concerning the part played by the vitreous after a retinal tear has been formed. The involved eye is always at least relatively hypotonic because, according to Leber, the intra-ocular fluids find a new outlet in the exposed choroid. This increased outflow, according to Lindner, calls for increased formation of fluid by the ciliary body. This fluid has the characteristics of regenerated, or second, aqueous and is assumed by Lindner to cause contraction of the vitreous. To prove this experimentally, Lindner performed on young rabbits what he calls a "vitreous-fistula operation." He exposed the sclera in the region of the equator, treated an area from 2 to 4 mm. in diameter after the method of Larsson, with nonperforating electrocoagulation, and trephined the sclera, choroid and retina with a 2 mm. trephine. The effects of the operation were a deepening of the anterior chamber, chemosis, edema of the lids and hypotony of the globe. All these changes started about three hours after the operation, reached their height after another three hours and persisted for about one week. Anatomically, the picture was that of acute hypotony in man, viz., a transudate filling the chambers, hyperemia of the uvea, an excessively deep anterior chamber and a partial or total serous retinal detachment.

Lindner interprets this picture as follows: After the trephining the intra-ocular pressure drops to zero. A rapid formation of aqueous rich in proteins sets in. This aqueous trickles through the vitreous toward the trephine hole and through the latter into the subconjunctival space. Under the influence of this regenerated aqueous a strong active contraction of the vitreous sets in, the retina and choroid become detached, and the lens is pulled backward.

The obvious objection to this interpretation is that the fistulation alone produces all the aforementioned changes and that there is no important change going on in the vitreous. To this Lindner replies that repetition of the operation on the same eye often fails to produce acute hypotony although at the operation a large amount of intra-ocular fluid escapes.

This operation performed on human glaucomatous eyes produced, as a rule, no acute hypotony—probably, according to Lindner, because the regenerated aqueous of human eyes is not rich in proteins. If, however, the operation was combined with subconjunctival injection of a 10 per cent solution of sodium chloride, it produced acute hypotony with choroidal detachment lasting a week. No permanent reduction of tension could be accomplished.

The spontaneous acute hypotony that occurs without apparent provocation in heretofore quiet eyes with retinal detachment Lindner explains

as caused by an iritis which suddenly increases the protein in the aqueous. The increased amount of protein makes the aqueous more capable of causing shrinking of the vitreous.

The contraction of the vitreous is discussed in the light of the recent article entitled "Structure of the Vitreous" by Friedenwald and Stiehler (*Arch. f. Ophth.* 14:789 [Nov.] 1935). Lindner's view of the contraction of the vitreous under the influence of regenerated aqueous can serve as a guide in the solution of clinical problems. For instance, in the treatment of retinal detachment it does not seem advisable, after the subretinal space has been emptied, to have a fistula form in the eye operated on. In detachment in an aphakic eye the regenerated aqueous can do more damage to the vitreous because of their proximity. Any lowering of the tension (by puncture of the anterior chamber or by iridectomy) makes the vitreous shrink temporarily. The tension-reducing effect of any inflammatory process (Hamburger) may be partly due to contraction of the vitreous.

P. C. KRONFELD.

Therapeutics

COD LIVER OIL AS LOCAL TREATMENT FOR EXTERNAL AFFECTIONS OF THE EYES. E. STEVENSON, *Brit. J. Ophth.* 20:416 (July) 1936.

In support of his conclusions, the chief of which is that cod liver oil has undoubted and rather surprising efficacy as a healing agent. Stevenson cites several cases, including cases of burns of the lids and eyeball and of the cornea, dendritic ulcer and relapsing keratitis, in which this agent was used with success. The notes on 150 patients treated at the clinic indicate that its use has greatly reduced the "pre-oil" number of necessary attendances. Its use should be withheld in the early stages of hypopyon keratitis. Cod liver oil applied locally seems to promote the growth of natural tissues and to inhibit the growth of scar tissue in any case in which there is loss of substance.

W. ZENTMAYER.

THERAPEUTIC EXPERIENCES WITH INHALATIONS OF AMYL NITRITE IN DISEASES OF THE FUNDUS. E. BIRÓ, *Klin. Monatsbl. f. Augenh.* 96:649 (May) 1936.

Biró reports on the experiments made with inhalations of amyl nitrite at E. von Grósz' ophthalmologic clinic at Peter-Pázmány University in Budapest during the last four years. The results of the treatment of divers ocular diseases are rubricated on a number of tables.

Vision was improved in 46 per cent of fifty-seven patients with chiefly chronic fundus changes. The improvement occurred mainly in hypertonic (nephritic) retinitis, choroiditis of nonsyphilitic origin, chorioretinitis, and recent and partial central embolism of the retina as well as in the dominantly hereditary type of pigmentary degeneration of the retina. No effect was recorded in locomotor atrophy of the optic nerve.

Small doses of amyl nitrite, limited to two or three inhalations, are free from risk if applied before meals under certain precautions. The

vascular system is examined with the tonoscillograph after the method of Plesch. The inhalations are not used in patients with brittle blood vessels in which the tonoscillograph shows wide excursions. It was observed that hemorrhages occurred in patients with hypertension of about 200 mm. of mercury as well as in some with normal blood pressure but wide excursions of the apparatus. The observation showed, in Biró's opinion, that hemorrhages are less the result of hypertension than of diseased blood vessels.

K. L. STOLL.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

March 19, 1936

CHARLES R. HEED, M.D., *Chairman*

A. G. FEWELL, M.D., *Clerk*

PRESENTATION OF THREE CLINICAL CASES. DR. WARREN S. REESE.

(a) *Syphilitic Optic Neuritis*.—A colored girl aged 16 stated that three weeks ago frontal headache developed and she noticed that she was losing vision of the left eye. Three days ago the right eye became red and painful. Vision in the right eye was 6/9, and in the left, perception of movements of the hand. External examination revealed episcleritis of the upper outer quadrant of the right eye and a pupil that did not react to light on the left side. The left fundus showed a markedly choked disk, and the swelling measured from 5 to 7 diopters. The macular region presented an incomplete star-shaped figure, while the periphery of the fundus was normal. Physical examination gave negative results except for chronic tonsillitis and adenoiditis. The Wassermann test of the blood and of the spinal fluid was negative. A second and a third Wassermann test of the blood were positive to the extent of four plus. The papilledema gradually subsided under antisyphilitic treatment, and on March 12 numerous fine vitreous opacities appeared in the left eye. The pains in the head, which were most severe at night, diminished, and vision began to improve.

(b) *Dermoid Cyst of the Globe*.—In the left eye of a girl, just beyond the outer limbus, was a growth that resembled a typical dermoid, except that it had no hair growing from it. The irregular and rather extensive cystic growth involving the upper nasal quadrant of the same eye appeared to be an unusual type of dermoid.

(c) *Pituitary Tumor*.—A woman aged 44 complained of a growth on the right eye which had been present for the past five weeks. Vision of both eyes was 6/60; it was improved to 6/9 in the right eye and to 6/12 in the left with a plus 3.25 diopter sphere. External examination gave negative results except for a pear-shaped cystlike elevation at the inner limbus of the right eye, measuring about 4.5 mm. in length and 2 mm. in width. Slit lamp examination gave negative results. Ophthalmoscopic examination revealed a condition which was thought to be pseudoneuritis, both disks being slightly elevated and their margins obliterated. The vessels showed no abnormalities. Perimetric examination gave negative results except for a markedly enlarged blindspot, especially in the left eye. Neurologic examination disclosed a rather marked metabolic disorder of the hypopituitary and hypo-ovarian type but no definite evidence of organic nervous disease. A roentgenogram showed erosion of the sella turcica, indicating an intrasellar growth.

DISCUSSION

DR. WILLIAM ZENTMAYER: The ophthalmoscopic appearance in the third case resembles that in pseudopapilledema, but the diagnosis cannot be based on this alone. The absence of both engorgement and enlargement of the retinal veins and the facts that the fields show no changes except enlargement of the blindspot and that papilledema is rare in cases of pituitary tumor leave the diagnosis open. I think that a correct diagnosis cannot be made unless the patient is followed up.

A CASE OF SYMPATHETIC OPHTHALMIA WITH SUBSTANTIAL RECOVERY OF VISION. DR. C. E. SHANNON.

G. H. P., a man aged 34, visited my office in October 1935, complaining of pain and tenderness in the left eye, associated with marked depreciation of vision. The history, which is somewhat fragmentary, is as follows:

In July 1935, while he was opening a box, his right eye was struck by a nail, which produced a penetrating wound of the cornea at 5 o'clock, with prolapse of the iris, and laceration of the crystalline lens. The patient was immediately sent to the hospital and given an injection of antitetanus serum and appropriate local treatment. Three days later the prolapsed iris was excised; the lens was extracted by the linear method, and a conjunctival flap placed. As a precautionary measure daily injections of 5 cc. of milk and large doses of sodium salicylate were given. Four weeks after the accident there was no evidence of sympathetic irritation or inflammation, although the injured eye was still slightly congested, and the patient was discharged. The Wassermann and Kahn tests were negative.

The patient was readmitted to the hospital about ten weeks after the initial injury, with a history of lacrimation and diminution of vision of the sympathizing eye, which had developed a week prior to readmission and approximately nine weeks from the date of injury. The exciting right eye was painful and congested and was enucleated at once. Unfortunately, no microscopic study of the eyeball was made. Four and a half weeks after the enucleation examination revealed a quiet right socket and a left eye moderately congested, which was tender on pressure. The left eye had vision of fingers at 7 feet (213.4 cm.). Slit lamp study demonstrated infiltration throughout the substantia propria, and precipitates on the posterior surface with many linear markings resembling folds in Descemet's membrane, and the iris showed many posterior synechiae, while numerous deposits of pigment were noted on the anterior capsule of the lens. The transilluminating beam of light was slightly relucant from the aqueous, and the pupil was irregular and moderately dilated. No details of the fundus were obtainable on account of the corneal infiltration and, probably, opacities of the vitreous. The tension, taken with the fingers, was apparently normal.

The patient was immediately sent to the hospital for active treatment, which consisted of the following measures: intravenous injections of typhoid vaccine every other day, beginning with a dose of 0.1 cc., representing 100,000,000 germs, which was gradually increased to 0.3 cc.; mercurial inunctions twice daily, and, as local measures, treatment with atropine, ethylmorphine hydrochloride and hot compresses. The reactions from the typhoid vaccine were satisfactory, the temperature

reaching approximately 102 F. after each injection. After the third injection the eye began to quiet down, and vision showed definite signs of improvement. The treatment was continued until the patient had received ten injections of typhoid vaccine. After three weeks of treatment the left eye showed vision of 6/6. The slit lamp showed that the infiltration of the cornea had almost entirely disappeared, but the deposits of pigment on the anterior capsule were unchanged. With the ophthalmoscope it was noted that the vitreous opacities had in a great measure been absorbed, and the details of the fundus were fairly clear, with no gross lesions.

DISCUSSION

DR. GEORGE DE SCHWEINITZ: I have in mind the case of a woman who had fully developed sympathetic ophthalmia, the result of a neglected laceration of the eye. She was sent to the Hospital of the University of Pennsylvania, where she received the treatment which was customary at this period, a number of years ago, and ultimately obtained a complete cure, but not until a low grade nephritis had yielded to treatment. I believe that it is essential in all cases of sympathetic ophthalmia to make a thorough general examination and to eliminate any general disease factor that might be discovered.

DR. H. MAXWELL LANGDON: Concerning the enucleation of the exciting eye, I think that a great deal of care should be taken before this is done. If the eye is blind it should be removed, as this may possibly do some good even if the sympathetic attack has started. If, however, the eye has vision which is at all useful it probably should not be removed, as after the sympathetic attack it may be the better eye of the two. This, of course, does not apply to eyes which may cause sympathetic disease before such an attack has started. In such cases removal is definitely indicated.

DR. J. MILTON GRISCOM: Dr. Jonas S. Friedenwald made the suggestion that exposure of the bodies of patients suffering from sympathetic ophthalmia to ultraviolet rays might result in stimulation of pigment in a locality distant from the eye, thus supplying an antigen which would combine with the antibodies. He has treated three patients by this method, with favorable results.

ANGIOID STREAKS OF THE RETINA ASSOCIATED WITH PSEUDOXANTHOMA ELASTICUM AND DISCIFORM MACULAR DEGENERATION.

DR. EDMUND B. SPAETH and DR. JOSEPH I. GOUTERMAN.

After a brief outline of the highlights of angioid streaks and pseudoxanthoma elasticum, a typical case is described. The patient was a colored woman 58 years of age. The characteristic ocular lesions consisted of bilateral circumpapillary rings (complete in the left eye and incomplete in the right eye), radiating angioid streaks on a slate-gray peripapillary zone, extensive retinal hemorrhages in various stages of absorption and bilateral disciform macular exudation. The clinical dermatologic lesions were pathognomonic of pseudoxanthoma elasticum, and a biopsy of skin clinched the diagnosis.

DISCUSSION

DR. SIGMUND S. GREENBAUM: Pseudoxanthoma elasticum is in no way, so far as is known, related to xanthoma. The latter condition is

an infiltrative process, particularly in the skin, in which it produces tumors, the result of degeneration of fat. There are about 16 reported instances of associated angioid streaks of the retina and pseudoxanthoma elasticum. This represents about 25 per cent of the reported instances of pseudoxanthoma elasticum, and one would infer that the two conditions may in some way be related. The cutaneous histologic features are perfectly clear: There is elastic degeneration in the dermic structures, and it may be that this represents a presenile change, since the same histologic picture is present in senile degeneration of the skin. In the past twenty years I have seen 3 instances of pseudoxanthoma elasticum: the first while I was a student in Paris and the second about nine years ago: the present case represents the third. In the first two cases no examination was made for angioid streaks, since the association was noted only some six or seven years ago. The present case is the second one in which the diagnosis of pseudoxanthoma elasticum followed the discovery of the presence of angioid streaks.

DR. WILLIAM ZENTMAYER: The patient, the fundus of whose eye was shown on the screen, was under my observation for twenty-five years. The last time that he was seen was in 1934, when he was reexamined at the request of Dr. Terry for evidence of osteitis deformans. He was thoroughly studied by Dr. Arnett, and roentgenograms of the skeleton were taken, but there was no evidence of Paget's disease.

The case reported by Dr. Spaeth and Dr. Gouterman is of special interest because of the association of the streaks with disciform macular degeneration. Batten has called attention to the frequency with which circinate retinitis, which is allied to disciform macular degeneration, is found in association with angioid streaks.

The subsequent history of Verhoeff's patient, as given by Dr. Terry, is that the halo about the papilla is more prominent, and black streaks are now noticeable. This, of course, would strengthen Verhoeff's assumption that the opposite eye, of which he made a histologic study, was the seat of angioid streaks.

In view of the facts that up to 1934 but 9 cases of angioid streaks in association with Paget's disease had been reported, that the examination of 22 patients with Paget's disease showed angioid streaks in 3, and that 23 cases of pseudoxanthoma elasticum with associated angioid streaks have been reported, I think that unless the two general conditions have a common pathogenesis there must be some underlying common cause for the cutaneous condition and the ocular condition.

April 16, 1936

CHARLES R. HEED, M.D., *Chairman*

A. G. FEWELL, M.D., *Clerk*

MARGINAL DEGENERATION OF THE CORNEA WITH PROLAPSE OF THE IRIS. DR. WILLIAM ZENTMAYER.

Dr. Zentmayer reviewed the recent literature on marginal degeneration of the cornea and discussed the clinical aspects, including the biomicroscopic findings and the histologic features of this condition. He

called attention to the fact that there are but few cases reported in the American literature and that in all the cases reported there were only 7 with prolapse of the iris as a complication.

On Oct. 21, 1935, Mrs. C. F., aged 52, was referred to Dr. Zentmayer by Dr. M. E. Smukler. The patient was born in Russia and came to America fourteen years before. There was a history of inflamed eyes during childhood. In 1930 she was under treatment for hyperthyroidism, and the same year Dr. Smukler did a multiple ignipuncture on the lower lid of the left eye for entropion and trichiasis. At about the time that Dr. Smukler first saw the patient she had consulted a druggist because of a foreign body in the right eye, and in removing it he noted the prolapse of the iris. In both eyes there was marginal dystrophy of the entire circumference of the cornea. The resulting furrow was of uneven depth. In the right eye there was a prolapse of the iris about 3 mm. in diameter through the floor of the furrow. The protruding iris tissue was covered by the epithelium. The sensibility of the cornea was subnormal. The epithelium was intact, as was evidenced by the absence of staining with fluorescein. The tension of the right eye was 22 mm. of mercury, and that of the left, 20 mm. A 1 per cent solution of physostigmine salicylate was instilled into the eye, with little or no effect on the size of the prolapse.

Biomicroscopic examination revealed that, as the corneal image of the light was swept across the limbus, it became constricted over the grooved portion. The constriction was particularly marked at the upper margin, where a deep groove was apparent, reaching well into the substantia propria. The epithelium was intact. The limbic vascular loops extended farther into the cornea than normally. It was impossible to determine whether the endothelium was intact in the involved portion of the cornea, but it was normal in the surrounding area. No folds were visible in Descemet's membrane. There were disseminated areas of thinning of the uveal pigment in the zone of the sphincter. The lens was normal.

DISCUSSION

DR. ALFRED COWAN: The slit lamp picture of the type of marginal dystrophy shown here by Dr. Zentmayer corresponds to the microscopic findings, as it seems that the condition is due to a loss of cornea proper, leaving both the epithelial and the endothelial surfaces intact, so that there is a gutter-like depression of the anterior surface, while the posterior surface remains intact. At the portion where the iris is prolapsed it is hard to tell with the slit lamp whether one is dealing with an epithelialized surface of the iris or whether the iris is covered with intact Descemet's membrane; but it seems that it is the surface of the iris and not Descemet's membrane which is seen over the hernia. It is easy to see in an ordinary descemetocle that the cornea is lost down to Descemet's membrane, but in this case, since the corneal propria is absent in the neighborhood of the ectasia, the anterior and posterior surfaces of the cornea become narrower toward the edge of the prolapsed iris. In those cases of corneal abscess in which the entire cornea sloughs off, leaving the bare iris, the surface of the iris becomes epithelialized in a short time and resembles the condition under discussion. However, in 2 cases mentioned by Koby, one of his own

and one of Trantas, in each of which there was an ectasia of the cornea, Descemet's membrane, from the description, seemed to remain intact in one and be perforated in the other.

COMPLETE REATTACHMENT OF THE RETINA FOLLOWING OPERATION WITH THE THERMOPHORE. DR. ANDREW KNOX (by invitation of Dr. H. Maxwell Langdon).

W. L., aged 52, entered the Hospital of the Protestant Episcopal Church on Jan. 27, 1936, complaining of marked depreciation of vision in the right eye of one week's duration. Examination with the ophthalmoscope revealed a number of small dark opacities located in the vitreous. There was a large detachment of the retina extending from 9 to 2 o'clock, with a marked dip in the center. Transillumination showed no abnormality. Vision was movements of the fingers above the point of fixation. There was complete loss of the visual field. The left eye was normal externally and intra-ocularly, with vision of 6/6.

The patient was admitted to the hospital and placed on his back, sand-bags being placed on each side of his head. Atropine was instilled into each eye, and pinpoint goggles were ordered. On the day after admission to the hospital the patient stated that he thought that his vision had improved slightly. On February 3 the detachment showed improvement, and there was some increase in the size of the field. On February 4 the patient was operated on by Dr. Langdon. The following operative procedure was carried out: After the eye was prepared in the usual manner, 3 drops of a 4 per cent solution of cocaine hydrochloride was instilled at three minute intervals. A retrobulbar injection of a 2 per cent solution of procaine hydrochloride was also given. The bulbar conjunctiva was dissected freely, well beyond the area of the detachment. The sclera was then pierced on either side of the superior rectus muscle with a von Graefe knife, thus allowing the subretinal fluid to exude. The thermophore was then applied seven times at different points on the sclera corresponding to the area of detachment. The thermophore at the temperature of 168 F. was applied to the sclera for one minute at each application. The conjunctiva was replaced and sutured, and the patient was returned to the ward. After forty-eight hours the eye was dressed and carefully examined. There was a small amount of edema of the lids and conjunctiva. Examination with the ophthalmoscope showed the retina to be completely reattached. The fields were roughly taken and seemed to be full. The patient was confined to bed for three weeks after the operation. The fields were again taken at the end of this time and showed practically a complete recovery. Vision was 20/40 with correction.

DISCUSSION

DR. H. MAXWELL LANGDON: This procedure is simple. One great advantage is that it does not disturb the eye, and if one does not get good results it is possible to go ahead with whatever operation one may wish. That this particular type of operation works has been proved, I think, by the 3 cases I have presented here: in one the operation was done in September 1934; in the second, last November, and in the third, in February. The patient who was operated on in September

1934 has still the same field and the same central vision, and there is no change whatsoever. The patient who was operated on in November has a full field, with vision of 5/22. The retina was detached four months before I saw her. If the patient had been seen earlier I think that better vision would have resulted. The operation is one, I believe, that any one could do, and I hope that some one will try it and see how it works in a series of cases other than the type my associates and I have observed.

TUBERCULIN TESTS IN OPHTHALMOLOGIC CASES. DR. GEORGE P. MEYER (by invitation of Dr. Francis H. Adler).

In the study of ocular tuberculosis a properly performed intracutaneous tuberculin or Mantoux test is of great assistance. It is important to observe certain precautions in the technic in performing the test and to use a potent old tuberculin or, preferably, the purified protein derivative. The latter product has the advantage of being stable and uniformly potent in its commercial tablet form. A positive Mantoux test indicates a previous tuberculous infection. It indicates that the patient is hypersensitive. It does not indicate presence or lack of activity in the lesion. The degree of hypersensitivity does not parallel the degree of immunity. A study is being made to determine whether patients with ocular tuberculosis manifest a higher degree of sensitivity than nontuberculous persons.

INTERPRETATION OF CASES OF UVEAL TUBERCULOSIS AND EXPERIMENTAL TUBERCULOUS ALLERGY. DR. FRANCIS H. ADLER.

This paper will be published in full in a later issue of the ARCHIVES.

HISTOPATHOLOGY OF EXPERIMENTAL TUBERCULOUS ALLERGY OF THE CHOROID. DR. ESMOND R. LONG (by invitation of Dr. Francis H. Adler).

Previous experiments have shown that the cornea in tuberculous guinea-pigs is exquisitely sensitive to tuberculin. In the experiments reported at this time the effects of the instillation of tuberculin into the uveal tract were studied. The purified protein derivative of tuberculin was used. Up to the present in these experiments efforts have been largely directed toward determining minimal doses effective in producing allergic reactions in the choroid. A dose of 0.000005 mg. of the purified protein derivative proved too small to produce cellular inflammatory reactions visible at the end of four days, although a cellular protein exudate between the choroid coat and the retina, which was not present in the control animals, followed the introduction of this dose in sensitive guinea-pigs in a number of cases. Somewhat higher doses were likewise ineffective in producing the well known type of cellular inflammation occurring in the skin and cornea of tuberculous guinea-pigs on suitable injection. On the other hand, an intense cellular inflammation of the choroid followed the subsecleral injection of 0.01 mg. of the purified protein derivative into tuberculin-sensitive guinea-pigs. Three days after injection the choroid was distended to five times the normal thickness as a result of infiltration by polymorphonuclear leuko-

cytes and monocytes. Evidence from other experiments suggests that the initial infiltration was with the polymorphonuclear leukocytes and that rapid replacement by the monocytes was occurring by the third day. In an effort to simulate tuberculosis of the choroid without the use of tubercle bacilli, experiments are in progress in which purified phosphatide from the tubercle bacillus, with or without the purified protein derivative of tuberculin, is instilled subsclerally. Definite tubercle-like nodules, tending to be absorbed in sixteen days, have been produced by this method in a number of experiments.

DISCUSSION

DR. H. MAXWELL LANGDON: There are two or three things of great interest in this splendid presentation. One is the number of negative responses which Dr. Meyer reported in the investigation carried out by his colleagues and himself. As he said, the ophthalmologist is surprised when he has a negative report, because he is led to believe that tuberculous infection is widespread. I should like to ask Dr. Long whether he thinks that over a long period there would be a great variation of sensitivity. Of course, I know that sensitivity would not vary in a few weeks or a month, but would it vary in about five or eight years?

The picture which Dr. Adler showed of a macular lesion from Dr. Wilmer's atlas has been of interest to me because I have a patient with an identical condition at present. The only demonstrable source of infection was two badly infected teeth on the side of the affected eye.

DR. ESMOND R. LONG: The conditions modifying the intensity of the tuberculin reaction are not all known. In the course of a long period the level of sensitivity may change for several reasons. The most intense reactions seem to occur in healthy persons in close and continued contact with open tuberculosis, that is, in persons who are resisting repeated infection. Hence changes in the intensity of the exposure may modify the intensity of the sensitivity. Altogether apart from this, irregular seasonal variations in the strength of the tuberculin reaction have been observed when the same person is tested at intervals for a year or more. Certain changes occur regularly in patients with tuberculosis. It may be added that standard tuberculin of constant strength is a necessity in determining these variations in sensitivity.

DR. FRANCIS H. ADLER: Dr. Langdon's interesting observation confirms the fact that these tuberculous allergic lesions have nothing characteristic about them. They do not differ in any respect from other acute inflammatory lesions of the choroid. This also serves to prove the point that one can never deduce the cause of a lesion in the eye from the fact that it disappears when some focus of infection is removed, as these allergic lesions clear up rapidly of themselves.

ZONULAR OPACITY OF THE CORNEA. DR. WILLIAM J. HARRISON.

A man aged 58, a hatter, was exposed daily to the influence of flying fur, hair and mercury vapor, in the close confines of a room, for a working period of thirteen years. The opacity embraced an area in each cornea from .1 to 2 mm. from the limbus on each side and 6 mm.

from the limbus below and just slightly above the midline. The calcareous infiltration seemed for the most part to be in Bowman's membrane, extending at different parts into the substantia propria but never as deeply as Descemet's membrane. The tension was 29 mm. of mercury in each eye. Vision corrected was 20/20 in the right eye and 20/30 in the left. Full campimetric and perimetric fields without scotomas were obtained. The blindspots and the fundi were normal.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

Hanover, N. H., May 23, 1936

JAMES J. REGAN, M.D., *President*

WILLIAM P. BEETHAM, M.D., *Secretary*

INFLUENCE OF SIZE DIFFERENCES OF THE OCULAR IMAGES ON THE HOROPTER. DR. W. HERSAU.

The following types of differences in size are discussed: (1) physiologic differences correlated with asymmetrical convergence, (2) artificial differences induced by glasses of different magnifications and (3) the pathologic differences that occur in aniseikonia. The separate influence of the first and of the second type on the horopter was investigated (a special study having been made in Tschermak's laboratory), and it was found that while the rotation of the frontal plane horopter effected with magnifying lenses was following absolutely the computed amount, the rotation induced by the physiologic difference in size in asymmetrical convergence was much less than was mathematically expected. This indicated that a compensation takes place with asymmetrical convergence. This compensation would tend to meet the argument which is based on a well known fact, that rather large differences in size between the ocular images accompany asymmetrical convergence and yet, apparently, cause little or no ocular trouble. This has been cited against the importance of differences in size in general (von Hess, von Rohr and Erggelet) and against aniseikonia in particular (Jackson and Ludvig).

CHANGES IN THE RELATIVE SIZES OF THE OCULAR IMAGES WITH ASYMMETRICAL CONVERGENCE. DR. KENNETH OGLE.

Evidence is presented from several types of experiments on the haploscope which establishes the existence of some type of mechanism that compensates for the differences in the size of the retinal images introduced when one looks to the right or to the left at near visual distances—for example, in reading. These experiments showed nearly complete compensation in the vertical meridian and partial compensation in the horizontal meridian. These results tend to explain the fact that the differences in size introduced by asymmetrical convergence do not interfere with reading.

CLINICAL ASPECTS OF ANISEIKONIA. DR. E. H. CARLETON.

The correction of aniseikonia is evaluated from the clinical point of view by discussing a statistical summary of 300 cases in which patients had obtained relief through iseikonic prescriptions given by the eye clinic at Hanover, N. H. In 47 per cent, or nearly one-half of the cases, there was no significant change in the refractive correction (less than 0.37 diopter), while in only 23 per cent of the cases did the change in refraction exceed 0.37 diopter. In 21 per cent of the cases the refractive errors were small (less than 1 diopter in the maximum meridian). Ten patients with emmetropia exhibiting aniseikonia were found. With reference to the relationship of anisometropia to aniseikonia, it is stated that 15 per cent of the total number of patients were isometropic, while in 20 per cent the condition of aniseikonia was opposite to what would be expected from the condition of anisometropia. To present a clearer picture of the various combinations of refractive conditions that occur in conjunction with aniseikonia, four successful cases which illustrate these interrelations are presented: (1) a case of iseikonic correction without change in the refractive correction; (2) a case of emmetropia in which aniseikonia was demonstrated and corrected; (3) a case of anisometropia in which the iseikonic correction was opposite to that expected; (4) a case of anisometropia in which the correction of aniseikonia was in the same direction to the expected condition if it was the cause of the inequality of the ocular images. This case was of particular interest because the patient obtained complete relief from headaches of the migraine type from which she had suffered for sixteen years. From clinical experience it is believed that a large percentage of persons with anisometropia should wear iseikonic corrections. Clinical results so far obtained warrant the statement that aniseikonia is an important factor in binocular vision and that a clinical investigation of this anomaly should be made in all cases of functional disturbance of binocular vision as well as in cases in which relief has not been obtained by the correction of the refractive errors.

APPLICATION OF THE AFTER-IMAGE IN THE INVESTIGATION OF SQUINT.
DR. A. BIELSCHOWSKY.

This article will appear in full in a later issue of the ARCHIVES.

CORRECTION

In the author's proof of the article entitled "A Method for the Correction of Entropion in Trachomatous Patients, with Particular Attention to the Esthetic Results," by Archimede Busacca, which was received after publication of the article in the November issue of the ARCHIVES, changes were made to correct errors made by the translator which were not observed by the author before the article was submitted for publication. These corrections have been made in the reprint, a copy of which may be obtained by writing to the publication office.

Book Reviews

The Intellectual Functions of the Frontal Lobe: A Study Based upon Observation of a Man After Partial Bilateral Frontal Lobectomy. By Richard M. Brickner, B.S., M.D., Assistant Professor of Neurology, College of Physicians and Surgeons; Attending Neurologist, Neurological Institute, New York. Price, \$3.50. Pp. 354, with 8 anatomic photographs. New York: The Macmillan Company, 1936.

"The frontal lobe is, by common consent, the most dominant part of the brain. Yet in spite of its supreme position in the guidance of life, it is the least understood and perhaps also the least studied structure of the body." Thus writes Tilney, in his preface to Brickner's book, which is the history—the only one on record—of a man from whom a large portion of each frontal lobe had been removed because of an extensive meningioma and who survived for several years, leading a fairly well adjusted life.

The patient, aged 40, a successful member of the New York Stock Exchange, with many friends and a satisfactory family life, began to suffer from headache, impaired memory and absent-mindedness. The symptoms grew worse for a year, at the end of which time he fell into a coma. He was then operated on by Dr. Walter Dandy at the Johns Hopkins Hospital, who amputated each anterior lobe from a vertical line just anterior to Broca's area. The portions of the anterior lobes removed measured, respectively, 7 cm. and 7.5 cm. in the antero-posterior diameter. Broca's areas, the rolandic areas and the anterior and middle cerebral arteries were not disturbed. A year after the operations the patient came under Brickner's observation. Throughout a second year he was carefully studied neurologically and psychologically, and his mental status was compared with that described by his family and friends as existing before his illness.

Neurologic examinations made repeatedly in the second postoperative year gave negative results, except for the finding of a fine tremor of the head and hands, complete loss of smell on the left side and an almost complete loss of smell on the right. The eyes were normal. Four attacks of generalized clonic spasm occurred, but these were without focal signs.

Lesions of the frontal lobes have long been known to cause definite changes in character. Holmes, in 1931, distinguished three types, "one characterized by apathy and indifference, one by depression, intellectual enfeeblement, automaticity and incontinence, and one by restlessness, euphoria, lack of concern over the gravity of the situation, irritability, childishness, 'Witzelsucht' and marked egoism."

A year after the operations efforts were made to reeducate the patient, and detailed notes were kept describing his behavior. His own spoken words were written down, and these records, together with the records of the psychologic tests and their interpretation, make up the bulk of the volume.

The symptoms observed in the patient's intellectual sphere that had an emotional coloring were due to impairment of restraint and con-

sisted of boasting, mild hostility and the expression of angry, aggressive and puerile impulses. The symptoms observed in the intellectual sphere that did not have an emotional coloring were failure to synthesize memory pictures, difficulty in fixing the attention and impaired capacity to differentiate between the important and the unimportant. Also, there was impairment of memory for recent events and, because of this, a lack of learning capacity. A further group of symptoms included impairment of judgment, slow cerebration and *Witzelsucht*.

The jocular waggishness or facetiousness which is perhaps the most striking symptom of lesions of the frontal lobe was termed *Witzelsucht* by Oppenheim in 1890, and this name has since then been in general use. Golla in 1931 suggested that "facetiousness is simply a conscious defensive reaction when the patient dimly realizes that he is suffering from an inability to understand and react normally." It has been likened to the flow of jocularity often found in persons in the uninhibited alcoholic state. Brickner expresses the belief that it, like many other symptoms of lesions of the frontal lobe, is due merely to impaired restraint. This symptom is of interest to the ophthalmologist because when he finds pressure atrophy of one optic nerve, with or without papilledema of the other, together with *Witzelsucht*, the diagnosis of a tumor in the frontal lobe on the side of the atrophy is at once suggested.

On assembling the many symptoms presented by his patient Brickner found that the personality had lost no characteristics and had gained none, but that some were more pronounced than before the illness and others less so. He states the belief that all the symptoms can be referred to a single intellectual deficiency, namely, an impairment of the process of synthesis.

In summing up, Brickner comes to a number of conclusions, from which the following statements are extracted:

A. While the patient's symptoms are numerous, none of them indicates an alteration in the fundamental nature of any mental process, but only the impairment of its completeness. Hence the changes are fundamentally not qualitative but are quantitative.

B. Only one function is considered as primarily affected. This is the elaborate association or synthesis into complex structures of the simpler engrammic products (latent memory pictures) associated in the more posterior parts of the brain. There is a diminution in the amount of this synthesis which places a limit on the degree of attainable complexity of thought. Through this deficiency a variety of defects become manifest, and the personality appears to be greatly altered.

C. While many of the symptoms have an emotional coloring, there is nothing to indicate an emotional disturbance in a primary sense.

D. The deduction seems justified that the frontal lobes are not intellectual *centers* in any sense except, perhaps, a quantitative one and that they play no specialized rôle in intellectual function. They add to intellectual intricacy in a quantitative manner only, by increasing the number of possible associations between engrams which have already been aggregated to a complex degree in other parts of the nervous system.

WARD A. HOLDEN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.

President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6^e).

Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ost-flandern, Belgium.

All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.

Place: Cairo. Time: December 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.

Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.

Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.

Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.

Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1939.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7^e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.

Secretary: Prof. A. Wagenmann, Heidelberg.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.

Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.

Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.

Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.

Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.

Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.

Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.

Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.

Place: Salt Lake City, Utah. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.

Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

Place: Seattle. Time: January 1937.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
 Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
 Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
 Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
 Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohi, Yankton, S. D.
 Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION. SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
 Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
 Secretary-Treasurer: Dr. Maurice C. Lorce, 120 W. Hillsdale St., Lansing, Mich.
 Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
 Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
 Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
 Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR,
 NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
 Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
 Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
 Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
 Secretary: Dr. Frederick V. Overman, 705 Hume-Mansur Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Burton W. Fassett, Geer Bldg., Durham.
Secretary-Treasurer: Dr. Casper W. Jennings, 332 N. Elm St., Greensboro.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in
October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervy Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
 Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
 Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
 Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
 Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
 Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
 Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
 EYE, EAR, NOSE AND THROAT

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
 Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
 Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
 Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
 Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
 Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
 Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
 Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
 Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
 Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
 Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
 Secretary-Treasurer: Dr. Meyer H. Riwehun, 367 Linwood Ave., Buffalo.
 Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.

Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.

Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.

Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.

Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.

Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.

Place: Athletic Club. Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.

Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.

Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.

Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.

Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.

Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.

Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
 Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
 Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
 Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
 Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
 Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
 Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
 Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
 Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
 Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
 Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William A. Boyce, 727 W. 7th St., Los Angeles.
 Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
 Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
 Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
 Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.

Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.

Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.

Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.

Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in alphabetical order.

Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.

Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.

Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.

Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.

Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.

Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Kate S. Zeffoss, 119, 7th Ave., N., Nashville, Tenn.

Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.

Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.

Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.

Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St. New York.

Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.

Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.

Time: Second Thursday of each month from October to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every
month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each
month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June,
July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Robert H. Courtney, Professional Bldg., Richmond, Va.
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ACETYLCHOLINE IN THE TREATMENT OF ACUTE RETROBULBAR NEURITIS

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NEW YORK

Owing to the fact that sodium nitrite administered intravenously and amyl nitrite given by inhalation have both been found to be very effective in aborting attacks of acute retrobulbar neuritis,¹ with resulting rapid improvement in vision, it was deemed advisable to study the action of acetylcholine, a powerful but little used vasodilator, in this interesting disease.

In 1931 de Saint-Martin² reported a case of retrobulbar neuritis in which he thought the condition was improved after the use of acetylcholine. A woman of 31 had noticed gradual failure in vision, which began six weeks after the birth of a child. Five months later vision was found to be 6/10 in the right eye and perception of light in the left eye. The visual fields were contracted, and there was probably a central scotoma in each eye. The fundi were normal. A submucous resection of the nasal septum was done, and subcutaneous injections of atropine sulfate (1 mg.) were given every other day. Vision improved to 8/10 in the right eye in eight days and to 4/10 in the left eye in one month. Three months later vision was 7/10 in the right eye and 4/10 in the left eye. At this time one of the turbinate bones was removed. Three weeks after this second nasal operation vision was unchanged; so alternating injections of acetylcholine chloride and atropine were given for six days, after which the atropine was discontinued. Six weeks later, after twelve injections of acetylcholine had been given, vision was 10/10 in the right eye and 8/10 in the left. This improvement was maintained for two months, during which time twelve more injections of acetylcholine were given. This case was complicated by the fact that

From the Herman Knapp Memorial Eye Hospital.

Read before the New York Academy of Medicine, Section of Ophthalmology, Nov. 16, 1936.

1. Duggan, W. F.: Use of Vasodilators in Retrobulbar Neuritis, *Arch. Ophth.* **16**:380 (Sept.) 1936.

2. de Saint-Martin: Concerning the Efficiency and Diagnostic Value of Vasodilators, Especially Acetylcholine, in Retinal Angiospasm, *Ann. d'ocul.* **168**: 102 (Feb.) 1931.

Saint-Martin did not limit himself to one type of treatment. However, his explanation is that the improvement which followed the first nasal operation was due to the use of atropine subcutaneously, since no improvement occurred after the second operation until vasodilator therapy was instituted.

In 1932 Villaret, Justin-Besançon, Schiff-Wertheimer and Gallois³ stated that acetylcholine should be of value in the treatment of retrobulbar neuritis but that they had no personal experience of its value in such cases. However, both Bonnefon⁴ and Orr⁵ have reported good results following its use in chronic retrobulbar neuritis (toxic amblyopia).

CHEMISTRY AND PHYSIOLOGY OF ACETYLCHOLINE

In 1934 Alles⁶ reviewed the physiology of acetylcholine and other derivatives of choline, and in 1935 Gaddum⁷ wrote a comprehensive survey of the biochemical properties of the same substances. Both these articles have been used in preparing this section of this report.

Acetylcholine may be regarded as the product of a reaction between choline and acetylchloride. The presence of the acetyl group in the choline molecule increases the pharmacologic activity of choline at least a thousandfold. Thus, Burn⁸ found that the intravenous injection of 0.001 mg. of acetylcholine into a cat caused an immediate fall in the systolic blood pressure from 100 to 35 mm. of mercury and that the blood pressure returned to normal in approximately ninety seconds. (This amount of acetylcholine corresponds to a concentration of approximately 1:100,000,000.) With this fall in the blood pressure it was also shown that there was a marked dilatation of the arterioles in the limbs of the cat.

Although acetylcholine is such a powerful vasodilator, it is rapidly hydrolyzed and inactivated in the presence of extracts of blood or of tissue by a specific choline esterase which has been isolated. At 40 C. acetylcholine is almost completely destroyed by human blood within fifteen seconds.

3. Villaret, M.; Justin-Besançon, L.; Schiff-Wertheimer and Gallois, J.: The Esters of Choline in Ophthalmology, *Arch. d'opht.* **49**:129 (March) 1932.

4. Bonnefon: The Value of Acetylcholine in the Treatment of Toxic Amblyopia, *Prat. méd. franç.* **12**:65 (Feb.) (B) 1931.

5. Orr, H. Campbell: Acetylcholine in Tobacco Amblyopia, *Brit. M. J.* **2**: 69 (July 11) 1936.

6. Alles, G. A.: The Physiological Significance of Choline Derivatives, *Physiol. Rev.* **14**:276 (April) 1934.

7. Gaddum, J. H.: Choline and Allied Substances, in Luck, J. M.: *Annual Review of Biochemistry*, Stanford University, Calif., Stanford University Press, 1935, vol. 4, p. 311.

8. Burn, quoted by Evans, C. L.: *Recent Advances in Physiology*, ed. 4, Philadelphia, P. Blakiston's Son & Co., 1930.

Physostigmine has a powerful inhibitory action on choline esterase, although this inactivation is slow and reversible. A probable theory of this inhibition has been evolved by Stedman and Stedman,⁹ who suggested that physostigmine, which is itself a stable ester, acts by combining with the esterase in the same way and by the same mechanism as the choline esters do, thus blocking the enzyme. Moreover, urethanes inhibit choline esterase, and since physostigmine has a urethane group it is probable, according to Krause,¹⁰ that it is the urethane group of physostigmine which combines with the choline esterase.

At present, pharmacologic methods are used in estimating the amount of acetylcholine in tissues; 0.000001 mg. can be detected qualitatively, and 0.001 mg. can be estimated quantitatively, with an error not greater than 10 per cent. The most specific and sensitive tissue for the detection of acetylcholine is the longitudinal muscle from the anterior end of the dorsum of a leech (*Hirudo medicinalis*) suspended in a salt solution which contains physostigmine. Less sensitive tests include the action of acetylcholine on the isolated auricle of the rabbit's heart, on the frog's heart, on the small intestine of the mouse or of the rabbit, on the voluntary muscles of the frog and on the blood pressure of the cat or its effect on a cat's voluntary muscle which has been sensitized by previous section of the motor nerve.

It is extremely probable that acetylcholine is liberated in the tissues by the action of certain nerves (especially the parasympathetic nerves), just as an epinephrine-like substance (sympathin) is liberated by stimulation of the sympathetic nerves, and that the effects obtained by stimulating autonomic nerves is due to the liberation of acetylcholine or sympathin. Conversely, the results following the injection of acetylcholine simulate the results obtained by stimulating a parasympathetic nerve (just as the results following the injection of epinephrine hydrochloride simulate those caused by the stimulation of sympathetic nerves), only the action is fleeting and evanescent except in the presence of physostigmine. These facts are the basis for the theory of the humoral transmission of nervous impulses, particularly with reference to the autonomic nervous system. At present there is no purely chemical evidence identifying the substance liberated by the parasympathetic nerves as acetylcholine, but a large mass of circumstantial evidence has been accumulated by the work of Loewi, Dale, Gaddum, Chang, Feldberg, Magnus and many others.¹¹

9. Stedman and Stedman, quoted by Gaddum.⁷

10. Krause, A. C.: *The Biochemistry of the Eye*, Monograph 2, Baltimore, Md., Johns Hopkins Press, 1934.

11. Footnotes 6 and 7.

RÔLE OF ACETYLCHOLINE IN OCULAR MECHANISMS

In 1931 Englehart^{12a} reported that after stimulation of the third nerve the vitreous of a rabbit's eye which had been treated previously with physostigmine contained a substance which slowed the heart of a cold-blooded animal. Normal vitreous had no such effect. Moreover, when both eyes were treated with physostigmine and one eye was exposed to light the vitreous of this eye was rich in this substance (*Vagusstoff*, or acetylcholine) while the eye kept in darkness contained little or none of this substance. (Rabbits do not have a consensual light reflex.) Englehart expressed the belief that such a reaction is comparable to the effect of the vagus on the heart and that it is a humoral mechanism. Also, when the iris and ciliary body were isolated from the oculomotor nerve they contained none of this reactive substance (acetylcholine), but when the third nerve was intact and was stimulated electrically this substance could be detected in the ciliary body and iris.^{12b} This author later reported^{12c} finding a substance in the aqueous which was similar in all respects to acetylcholine when experiments similar to those just described were carried out, it being necessary, of course, to use physostigmine first to prevent the rapid disappearance of the substance.

In 1932 Velhagen^{13a} reported finding a substance similar to acetylcholine in alcoholic extracts of the uvea and retina as confirmed by its ability to contract the pupil and lower the intra-ocular tension. In 1933 he showed^{13b} that acetylcholine was able to constrict the isolated sphincter iridis in a dilution as high as 1:4,000,000, the effect being abolished by a previous application of atropine. In 1935, using the dorsal muscle of a leech which had been sensitized by the previous application of physostigmine, he demonstrated the presence of acetylcholine in the retina and uveal tract.^{13c}

According to Krause,¹⁰ both choline and acetylcholine are found in the normal moist bovine retina. It is assumed that they are formed in the retina from an unknown precursor as a step in the photochemical processes of vision.

In 1930 Duke-Elder^{14a} reported that the extra-ocular muscles contract *in vivo* following the intravenous injection of acetylcholine and

12. Englehart, E.: (a) The Mode of Oculomotor Stimulation, *Klin. Wchnschr.* **10**:26 (Jan. 3); (b) 215 (Jan. 31) 1931; (c) The Humoral Mechanism of Stimulation of the Third Nerve, *Arch. f. d. ges. Physiol.* **227**:220, 1931.

13. Velhagen, K.: (a) Intra-Ocular Substances Like Acetylcholine, *Klin. Monatsbl. f. Augenh.* **88**:846, 1932; abstr., *Arch. Ophth.* **9**:493 (March) 1933; (b) Experiments on Isolated Iris Muscles in Pharmacologic Research, *Arch. f. Augenh.* **107**:172, 1933; (c) Further Experiments on Active Principles in Ocular Tissues, *ibid.* **109**:195, 1935.

14. Duke-Elder, W. S.: (a) New Observations on the Physiology of the Extra-Ocular Muscles, *Tr. Ophth. Soc. U. Kingdom* **50**:181, 1930; (b) *Text-Book of Ophthalmology*, London, Henry Kimpton, 1932, vol. 1

in vitro ^{14b} when this substance is added to a muscle bath containing physostigmine. This action is unaffected by atropine, increased by epinephrine and abolished by both nicotine and curare. A similar reaction occurs in the normal striated muscles of species below the mammals and in the muscles of fetal mammals but in the voluntary muscles of fully developed mammals only after they have been deprived of their motor nerve supply. "At present, the significance of this reaction is not clear. It would seem that these muscles (extra-ocular) are able to avail themselves of an archaic and primitive mechanism which the other voluntary muscles have lost and which is analogous to that evident in the involuntary muscles" (Duke-Elder ^{14b}).

This brief summary will serve as an introduction to the use of acetylcholine in the treatment of acute retrobulbar neuritis. It must be emphasized that its use in this condition is dependent only on its vasodilating property. Moreover, the action of acetylcholine as a vasodilator is probably very transient, owing to its rapid inactivation by the choline esterase present in the blood and tissues. This characteristic, while probably decreasing its efficiency, adds a factor of safety to its use.

REPORT OF CASES

The two cases of acute retrobulbar neuritis which were the occasion for the present article are described briefly.

CASE 1.—C. P., a man of 31, was seen on Jan. 8, 1934, with the complaint of blurred vision in the right eye, which had been present for approximately two months. A tonsillectomy on Nov. 17, 1933, had been followed by a slight temporary improvement in vision; on December 18 vision was 20/30. There was no history of a recent cold in the head, and the patient did not use tobacco or alcohol.

Vision in the right eye (corrected) was 20/50 on Jan. 8, 1934, and 20/40 on January 12. The fundus showed hyperemia and nasal blurring of the disk, with engorgement of the retinal veins. On the tangent screen there was an oval scotoma 2 by 5 degrees in size just temporal to the fixation point for a 1 mm. white test object at a distance of 1 meter, and a 5 mm. red test object at a distance of 1 meter was identified only in a crescentic area in the nasal field. Vision in the left eye (corrected) varied between 20/30 and 20/20 on several occasions; the field was normal, and the fundus was similar to that of the right eye except that the retinal veins were not engorged. Both eyes had a high degree of compound hyperopic astigmatism. The Wassermann reaction of the blood was negative; there were no signs of multiple sclerosis, and the sinuses were normal on clinical examination.

This patient received three intramuscular injections of acetylcholine (0.1 Gm.) between January 12 and 19. On the tenth day after this treatment was begun (January 21) vision in the right eye was 20/20—, the scotoma was barely perceptible and the field for red was markedly improved. The fundus showed no change. This improvement was maintained for the next ten days, during which time two more injections of acetylcholine were given.

On Nov. 13, 1936, Dr. W. F. C. Steinbugler informed me that vision in the right eye was 20/20— and that the patient was not aware of any improvement in

vision until sphenoid sinusitis (diagnosed by the roentgen rays) had been treated. He received this treatment some time after Jan. 31, 1934. His failure to attribute any improvement to the injections of acetylcholine was probably due to the fact that he still had a small scotoma when he was last seen; he received injections every three days instead of daily as in case 2.

CASE 2.—R. C., a married woman of 21, was seen first on Dec. 2, 1935, with the history that four days before she had noticed marked blurring of the left vision on awakening. As she had been nursing a baby for three months the case might be classified as one of retrobulbar neuritis associated with lactation. However, a severe cold in the head developed on the day after her vision failed, which might also have been an etiologic factor.

Vision in the left eye was 8/200; the media were clear, and examination of the fundus revealed some blurring of the disk, with engorgement of the retinal veins. There was a dense scotoma about 30 degrees in diameter, including both the blindspot and the fixation point, for a 5 mm. white test object at a distance of 1 meter. A 10 mm. red test object at a distance of 1 meter was not identified in any part of the field. Vision of the right eye (corrected) was 20/25, and the field was normal. Both eyes were highly astigmatic.

Daily intramuscular injections of acetylcholine were instituted at once. On the fifth day after they were begun (December 6) immediately after the fifth injection vision in the left eye was 20/25 +. A small cecocentral scotoma for a 1 mm. white test object at a distance of 1 meter was present, and a 10 mm. red test object at a distance of 1 meter was perceived normally, except in the region of the cecocentral scotoma.

One month later vision was unchanged, and the field was completely normal. It was impossible to persuade this patient to have either a neurologic or a rhinologic examination.

The cases of eight patients suffering from attacks of acute retrobulbar neuritis who were treated with intravenous injections of sodium nitrite or with inhalations of amyl nitrite (one case) have been reported previously.¹ One patient was not improved; the other seven patients attained their best final vision in an average time of eight and one-half days.¹⁵ The two patients treated with acetylcholine obtained their best final vision in ten days and five days, respectively, the average time being seven and one-half days. Although only two cases are presented in this report, it must be evident that all these patients were treated only with vasodilators, so the results obtained in the patients treated with acetylcholine confirm the work done earlier with sodium nitrite and amyl nitrite.

SUMMARY

The chemistry and physiology of acetylcholine have been briefly reviewed.

The rôle of acetylcholine as a fundamental factor in the mechanism of the action of the parasympathetic (cholinergic) nerves has been outlined.

15. The time of improvement includes both the day on which treatment was begun and the first day that the best final vision was attained.

The relation of acetylcholine to normal ocular mechanisms has been briefly reviewed.

In the treatment of acute retrobulbar neuritis the vasodilating property of acetylcholine is of paramount importance.

CONCLUSIONS

Two patients with acute retrobulbar neuritis were treated only with acetylcholine injected intramuscularly; vision improved from 20/40 to 20/20—in ten days in the first case and from 8/200 to 20/25 in five days in the second case.

These results compare favorably with those obtained in eight other cases in which only intravenous injections of sodium nitrite, inhalations of amyl nitrite or a combination of the two was used.

The concept that acute retrobulbar neuritis is due to a vascular spasm in the optic nerve is relatively new and is based only on therapeutic results. As the treatment is simple for both the physician and the patient, it deserves further investigation by ophthalmologists before its actual value can be definitely evaluated.

DISCUSSION

DR. WILLIAM F. C. STEINBUGLER: I had occasion to see the patient first reported on and observed him for a period of four years before he came to me in November 1933 with the history of blurred vision of the right eye of short duration. I then made a diagnosis of retrobulbar neuritis and referred the patient to a rhinologist, who promptly removed his tonsils. After that the patient was transferred to the Herman Knapp Memorial Eye Hospital, so that I cannot report the effect that the injections had on his vision. At Dr. Duggan's request I examined the patient several days ago; vision then was 20/20 in the right eye and 20/30 in the left eye. The patient is positive that he obtained no lasting improvement until he had treatment for his sphenoid sinus, some time early in 1934.

DR. S. A. AGATSTON: I am puzzled about the vascular aspect of retrobulbar neuritis, because there is no definite proof that retrobulbar neuritis is caused by vascular spasm. Dr. Duggan seems to think there is a spasm of an artery in the optic nerve and stated in both of his case reports that the first discoverable sign was engorgement of the retinal veins. That in itself would indicate that there could not have been a spasm of the retinal artery, because in spasm of the retinal artery there is no engorgement of the veins. In retrobulbar neuritis or in any condition that causes a great deal of congestion of the optic nerve there is pressure on the vessels, the first effect being engorgement of the veins and the second effect, compression of the arteries, rather than spasm of the arteries. I do not deny the possibility that the vasodilators may effect improvement of the circulation mechanically. My only objection to the theory is that I do not think that the changes in the vessels can be caused by retinal arterial spasm.

TRAUMATIC GLAUCOMA

AN ANATOMIC AND CLINICAL STUDY

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I. ANATOMIC PART

Anatomic descriptions of traumatic glaucoma are rare, as only two cases have been published (Garnier [Garnye],¹ 1891; Morax, 1922). To this small number a few other cases may be added in which the eyeball had to be removed after contusion.

To exclude the possibility of intra-ocular infection clouding the picture, cases in which there was a complete rupture of the sclera or a perforating wound were not included in this study.

CASES REPORTED IN THE LITERATURE

CASE 1 (Garnier, 1891).—The globe of a boy aged 15 was removed fifteen days after an accident.

The sclera showed staphyloma medially and superiorly near the equator. The anterior drainage channels were normal. The pectinate ligament showed open clefts. Schlemm's canal was wide and open. The iris was hyperemic. Otherwise there were no pathologic changes; no displacement, no synechiae and no sclerosis. Examination of the lens revealed no dislocation and no cataract. The zonule was torn in one place. The ciliary body showed edema; the processes were larger and branched more markedly than normally. The ciliary epithelium was swollen and depigmented in many places by maceration; the bundles of muscles were pressed apart by fluid, and many muscle fibers were turbid and opaque. At the level of the tear of the zonule the radial muscle fibers were torn over a distance of about 1 mm. The large circular artery had also been torn and had thrombosed; the hemorrhage caused by this tear had been resorbed. The choroid showed marked hyperemia and edema everywhere; the suprachoroid was filled with fluid, especially at the equator, so that it had attained from five to six times its normal width. Examination of the sclera showed that the lymph channels in the sheaths of the blood vessels and the nerves were filled with pigment cells which had a lighter color than the choroidal cells; these cells were derived from the choroid and had been loosened by maceration. The optic nerve showed a mass of young connective tissue anterior to the optic papilla, perhaps a remnant of a previous hemorrhage.

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1. The spelling "Garnye" rather than "Garnier" is given first in the bibliography, since the article cited appeared in a Russian journal and the form "Garnye" is used in the Russian literature and in references to it in standard indexes.

In this case the pectinate ligament, Schlemm's canal, the angle of the anterior chamber and the iris showed no pathologic changes. The lens had not been dislocated. The depigmentation of the ciliary epithelium, which the author explained by maceration, had probably been caused by the trauma, together with the tear of the ciliary muscle and the rupture of the artery. The edema and hyperemia of the ciliary body and the accumulation of fluid in the suprachoroidea may be accepted. As to the accumulation of pigmented cells in the lymphatics around the blood vessels and the nerves in the sclera, one is inclined to think that these were chromatophores, which are normally present in all the sheaths and which have a lighter color than the pigment cells from the choroidal epithelium. It seems unlikely that the author has seen the same pigment granules that I have seen in the same place, as he clearly mentioned cells, not granules.

CASE 2 (Morax, 1921, eighteenth case).—An eye of a patient aged 65 was removed four weeks after an accident. The lens was subluxated anteriorly, with rupture of the zonule below. An albuminous, hemorrhagic exudate was seen in the anterior chamber. The iris was unrecognizable; only a few tracts and masses of pigment remained below, and rests of the pigment layer were adherent to the cornea above. At the periphery the iris lay against the cornea. The insertion of the ciliary processes seemed to be displaced backward. There was hemorrhagic infiltration of the lower half of the choroid. The outer layers of the retina were pathologically changed; the pigment epithelium had disappeared; the rods and cones were unrecognizable, and hemorrhages were seen in the layer of rods and cones and in the inner layers of the choroid. The inner granular layer and the layer of nerve fibers were in comparatively good condition and showed no hemorrhages. There were no signs of inflammation.

Although this eye showed a slight dislocation of the lens, Morax considered that this could not have been the cause of glaucoma. Morax also described a case of traumatic glaucoma in which the lens had been expelled through a rupture of the sclera. Obviously, the lens could not have caused glaucoma in that case, and no signs of inflammation could be observed.

CASE 3 (Lamb, 1927).—An eye of a patient aged 9 years was removed eight weeks after an accident.

The sclera was thin at the equator. The anterior chamber was full of blood, and the trabeculae of the filtration network were filled with red blood corpuscles. The iris was atrophic and about half the normal thickness. There was moderate proliferation of the chromatophores. The blood vessels were reduced to thin tubes, the majority even to capillaries. The number of muscle fibers of the sphincter muscle was diminished. The amount of albumin in the fluid in the stroma was increased. The posterior epithelium was thickened in some places, and there was loss of pigment in other places. In other places the epithelium was thin and interrupted. Here and there posterior synechiae were seen. The base of the iris had been torn through the pectinate ligament; the cleft that had thus been formed continued backward into the ciliary body just anterior to the ciliary muscle; much

blood was present in this gap, as well as in the surrounding tissue. The muscle of the ciliary body was thin; in the vascular layer many newly formed capillaries were full of polymorphonuclear leukocytes; in addition, a considerable number of plasma cells and proliferation of the chromatophores were seen here. The lens showed loss of anterior epithelium in the pupillary area and liquefaction of the fibers just behind this area. Outside the pupillary area a layer of fibers of the superficial part of the cortex containing vacuoles was seen; it ended behind the equator. The choroid was atrophic in its anterior and middle parts; many newly formed capillaries and proliferation of chromatophores were present. The retina showed loss of pigment of the pigment epithelium, cystic degeneration in its anterior half and marked proliferation of neuroglia cells in the posterior part. There were no pathologic changes of the optic nerve.

Lamb noted pathologic changes that corresponded in several respects to the changes that I observed (which will be described later), e. g., the changes in the iris, the rupture of the base of the iris into the ciliary body and also, perhaps, the changes in the choroid at the equator.

CASE 4 (Priestley Smith, 1881).—An eye of a patient aged 57 years was removed fifteen weeks after an accident. No blood was present in the eye. At the periphery the iris lay against the posterior surface of the cornea; there was coredialysis of 6 mm., and the iris had been torn completely from its base. The lens did not touch the ciliary processes. The ciliary muscle was perhaps somewhat atrophic. The optic nerve showed commencing excavation.

CASE 5 (Priestley Smith, 1883).—An eye of a patient aged 59 years was removed fifteen weeks after an accident.

In this case iridectomy was performed, after which the eye had to be removed immediately because of the enormous loss of vitreous. Large, darkly pigmented cells were scattered throughout the iris and its base. The angle of the anterior chamber was closed over a considerable distance. Schlemm's canal was partially filled with pigment; in some of the sections it was filled with erythrocytes. The lens showed rupture of the zonule medially and superiorly. The lens touched the ciliary processes. The relative positions of the iris, the ciliary processes and the lens were unreliable because of the operation, the loss of vitreous and the large hemorrhage. A large hemorrhage was seen outside the choroid and against the sclera. The optic nerve showed a deep but incomplete cup. The iris and the ciliary muscle were perhaps changed, but the difference between them and the normal structure was not obvious.

It is interesting to read that Priestley Smith observed that the iris and the ciliary muscle were normal or, at any rate, saw no obvious changes. The pigment granules in Schlemm's canal are best explained by a rupture of this canal.

CASE 6 (Leslie Buchanan, 1907).—An eye of a patient aged 59 years was removed six months after an accident.

Only the anterior half of the globe was examined. Rupture of the pectinate ligament and rupture of the inner layers of the sclera on the medial side were seen. Probably this represented a typical partial rupture of the sclera, originating in Schlemm's canal. In addition, ectasia was present at the equator, but its structure was not described in detail.

The most important of all these reports is Garnier's description. This author stated that the iris, as well as the angle of the anterior chamber, was normal, while the observations in his case bear a close resemblance to the microscopic observations in my cases.

REPORT OF TWO ADDITIONAL CASES

In two cases observed by me the globe was cut into serial sections. Every tenth section was then stained with iron hematoxylin or according to Van Gieson's method. Some of the intermediate sections were prepared later; whenever it was necessary, as in case of vessels, a complete series was examined. Special staining methods were employed to demonstrate the myelin sheaths of the nerves and to demonstrate the iron in the hematogenous pigment.

CASE 1.—Clinical History.—A. J. K., a shop assistant aged 17, stumbled and struck his left eye against the corner of a wooden box. He was examined the same day (Feb. 18, 1926). The right eye was normal and emmetropic, with visual acuity of 1.

The left eye showed a hematoma and superficial abrasions of the upper lid. There was extensive erosion of the corneal epithelium and of the bulbar conjunctiva. The anterior chamber was deep. The pupil did not react to light, and red light was reflected from the retina through the pupil. The tension was normal.

On the third day the patient had an intra-ocular hemorrhage and was admitted to the clinic.

On March 13 the tension was doubtful. On March 20 it was $+1$, and the corneal epithelium was vesiculated. On March 29 the tension was $+2$. On April 1 the globe was removed, as the sclera began to bulge superiorly.

Macroscopic Examination.—Even macroscopically the sclera was seen to be thin superiorly (fig. 1).

Microscopic Examination.—Cornea: The epithelium was irregularly thick. Its surface was rough; the nuclei of the cells were stained irregularly, and the cells were swollen. Here and there the epithelium had been lifted from Bowman's membrane to form vesicles of varying size.

Sclera (fig. 1): The sclera showed two ruptures of its inner layers—a typical rupture originating in Schlemm's canal superiorly and medially and an atypical rupture near the equator. These have been fully described elsewhere (Tillema, 1936).

Vessels in the Sclera: In the region of the incomplete rupture of the sclera Schlemm's canal had been destroyed. The veins of Schlemm's plexus were wide. They were surrounded here and there by small round cells and, especially below, by pigment granules containing iron.

Superiorly the wall of a ciliary artery had been disorganized (fig. 2). The lumen of the vessel was filled with a fine granular material, and its endothelium seemed swollen. The surrounding sclera showed the changes that have already been described elsewhere and which represent ruptures of the scleral fibers. Somewhat farther along blood pigment was seen in the wall of the vessel and in the sclera around the artery. The anterior branch running in the ciliary body was seen to be damaged, while the posterior branch had been obliterated and organized.

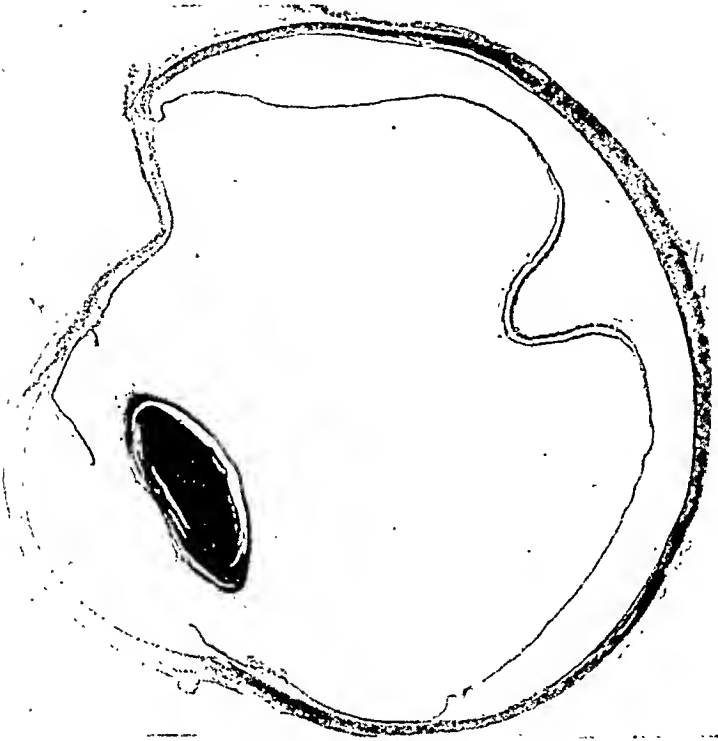


Fig. 1 (case 1).—General view of the injured eye. Note the paleness of the sclera above (incomplete, atypical rupture) and its thinness near the angle of the anterior chamber (incomplete, typical rupture). Also note the asymmetry of the anterior chamber.

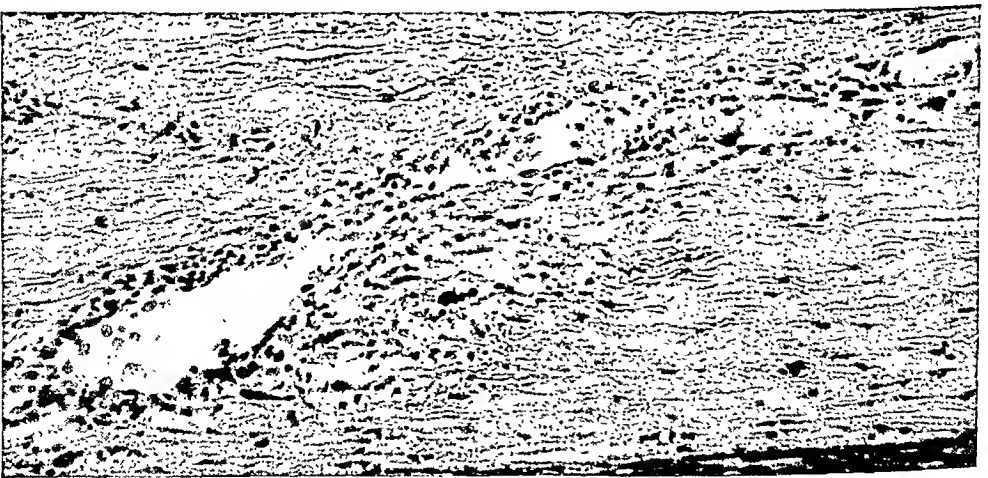


Fig. 2.—A ciliary artery. The wall of the vessel is partly disorganized. The surrounding sclera is very irregular in structure and contains scattered granules of both uveal and hematogenous pigment. Below, the ciliary body is just visible.

Another artery, on the inner nasal side, showed identical changes plainly. While it was intact in the outer layers of the sclera, its front wall in the inner layers of the sclera consisted of endothelium only (fig. 3), and a little farther it even showed a defect.

Analogous changes were seen in many other places in the anterior ciliary vessels, both in arteries and in veins.

The short posterior ciliary arteries showed no changes. One of the long posterior ciliary arteries, on the other hand, was seen to be interrupted in the inner

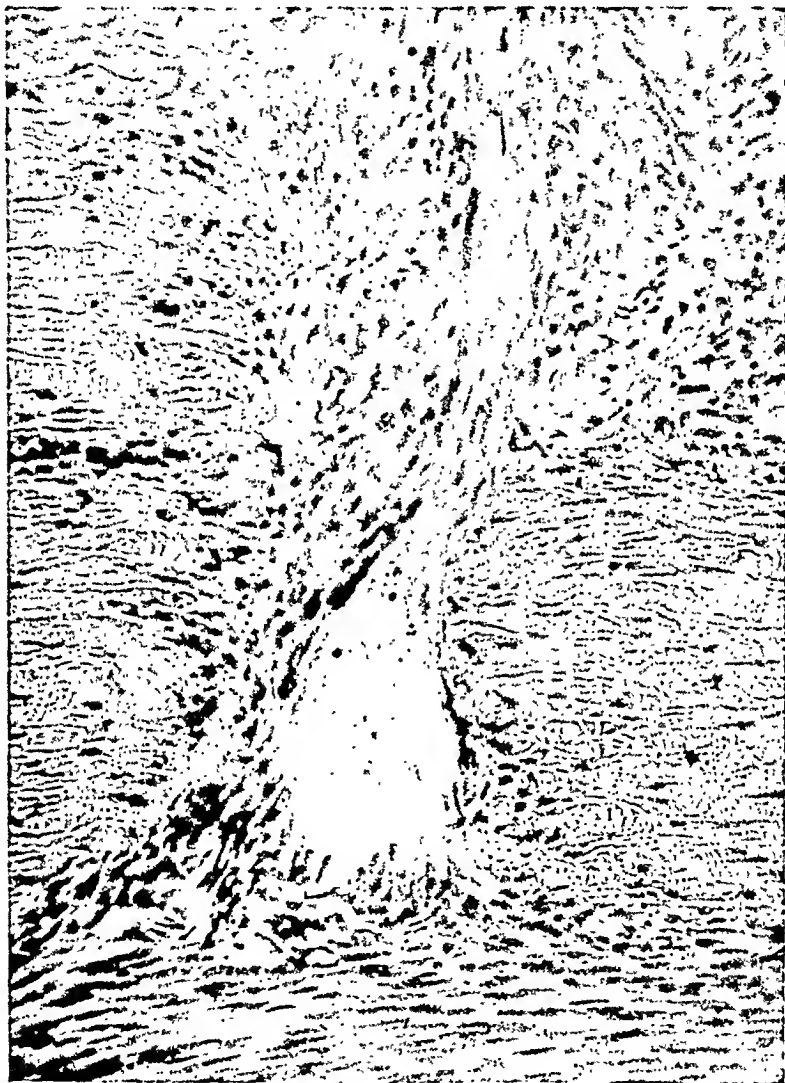


Fig. 3.—An anterior ciliary artery, showing on the right a strikingly thin wall. The wall shows a defect on the right and below. The ciliary body is seen below.

layers of the sclera. While it was well preserved in the outer layers of the sclera, with its accompanying nerve, in the inner layers of the sclera only an irregular mass of pigment granules was seen close to the nerve (figs. 4 and 5), but no vessel.

Five vortex veins were noted in the sections. The inferior nasal vein showed endothelial defects, which were covered with clustered white blood cells containing round and segmented nuclei. The surrounding fibers of the sclera ran a slightly irregular course. A few small round cells were scattered between them. Close to

the choroid the wall of the vessel was infiltrated by small round cells. Between the cells pigment containing iron was scattered. Slightly deeper in the choroid the wall of the vessel and the surrounding sclera contained a striking number of small round cells. Between and inside the endothelial cells of both the superior and the inferior temporal veins pigment granules were seen in a few places (fig. 6). The

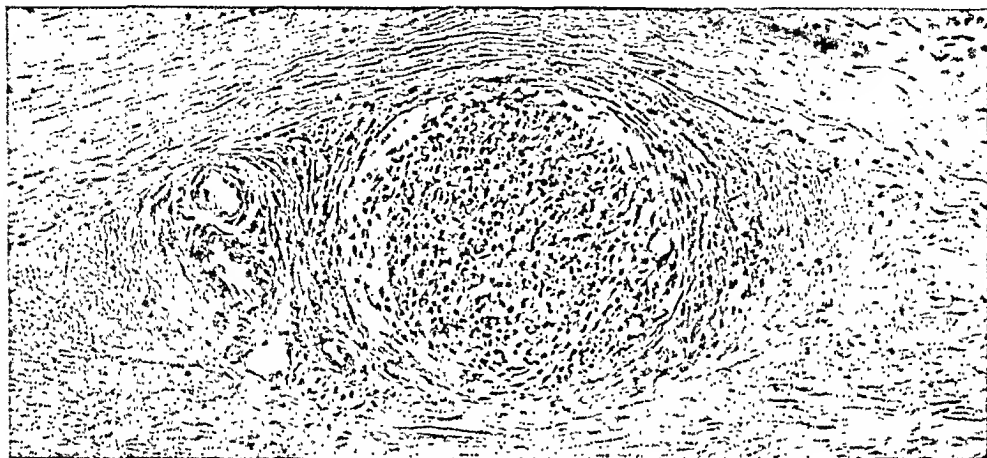


Fig. 4.—Posterior ciliary vessels and nerve in the outer layers of the sclera. Note the normal structure of all the tissues and compare with figure 5.

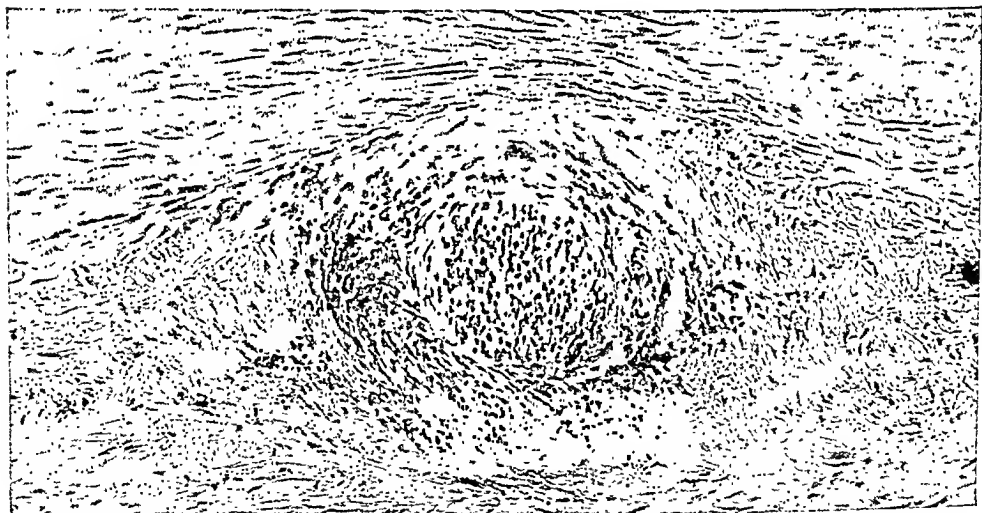


Fig. 5.—Same vessels and nerve as in figure 4. The vessels have been replaced by pigment (this is a section through the inner layers of the sclera, about 0.8 mm. from the section shown in figure 4).

other three veins were normal. However, pigment granules were observed in the perivascular channels of all five veins.

Ciliary Nerves: The ciliary nerves in general showed good structure and good staining properties. In a few instances, however, isolated groups of myelin sheaths were found to have degenerated, while the rest of the bundle was well preserved.

Anterior Chamber: The anterior chamber was remarkably deep. As the distance from the end of Descemet's membrane to the anterior ciliary processes was one and one-half times as large superiorly as inferiorly, the sclera must have been stretched in the same region (incomplete rupture of the sclera; see this section). The cornea, therefore, had been displaced forward. This explains the unusual depth of the anterior chamber. It also explains why the anterior chamber was deeper superiorly than it was inferiorly (fig. 1).

The iris had closed the angle of the anterior chamber everywhere. It was adherent to the cornea. The new-formed angle contained a small amount of granular material inferiorly. Superiorly the pectinate ligament had been greatly stretched, and some of the trabeculae were interrupted. Between the trabeculae many pigment granules were seen, and small round cells were noted. This pigment was largely hematogenous.

Iris: The anterior surface was torn superiorly. The stroma had lost a great deal of its normal structure in all its parts. It had taken the stains poorly, and

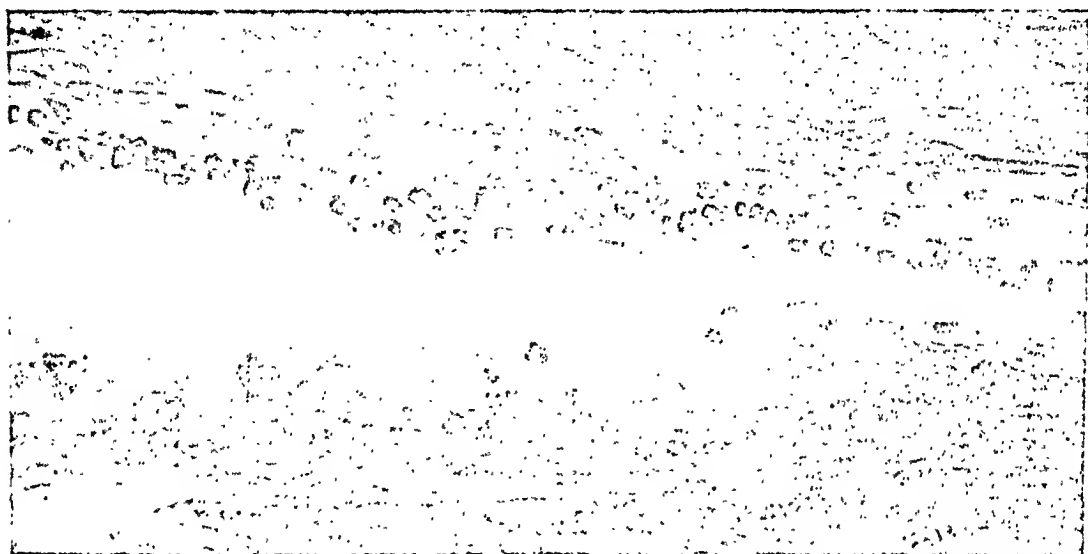


Fig. 6.—A vortex vein, showing pigment granules inside the endothelial cells.

it contained a great number of scattered lumps of pigment which were mostly hematogenous. The nuclei of the cells were pale, shrunken or disintegrated. Especially below, the stroma was almost structureless and had been replaced by a material that stained almost uniformly with eosin (necrosis of the iris; fig. 7). Below, near the ciliary body, the pigment in the posterior portion was scattered, mainly into the posterior chamber, but also into the stroma of the iris. A few giant cells with multiple nuclei were observed here.

The base of the iris was thin everywhere. It consisted almost exclusively of pigment epithelium. Inferiorly the iris had been split off the ciliary muscle. The new-formed angle of the anterior chamber continued backward into the ciliary body and contained a large amount of hematogenous pigment.

Ciliary Body: At the ora serrata the transition of the pars plana into the choroid had been destroyed. The changes seen in this region corresponded to those which will be described in the section on the choroid.

Inferiorly the ciliary processes had been partly destroyed. A large amount of pigment was lying scattered in the tissue of the ciliary body, partly in big lumps

and partly in fine granules. These fine granules were in part hematogenous. Here and there the pigment was arranged in layers, probably filling tissue spaces (necrosis of the ciliary body). Here and there, between the ciliary processes, small bundles of elongated cells were seen. It was difficult to decide whether these were fibroblasts or cells stretched by traction and belonging to the ciliary epithelium.

The pars plana of the ciliary body was separated from the sclera by a space containing fluid. Inferiorly this cleft was much more extensive than above, and it continued in the suprachoroidea as far back as the equator. In a lateral direction it was observed in nearly all the sections.

Choroid: Nasally and above, the choroid had been almost completely depigmented. In this region it was composed of loose cells and lumps of pigment



Fig. 7.—The iris, showing necrosis, with loss of structure and pigment granules scattered throughout the stroma. The angle of the anterior chamber is closed.

interspersed with spindle cells and a few small round cells (rupture of the choroid; fig. 8).

It has been mentioned already that the fluid-containing space between the ciliary body and the sclera continued backward outside the choroid (fig. 8) as far as the equator. Posteriorly also, however, the suprachoroid contained fluid, but only a small amount. Close to the optic papilla, especially laterally, the capillary layer of the choroid and the layer of middle veins had been replaced by closely packed spindle cells the nuclei of which stained well. These cells had broken through Bruch's membrane, and they were surrounded in the choroid by groups of small round cells (scar of a circumpapillary rupture of the choroid).

Lens and Zonule: The capsule of the lens was intact but slightly wrinkled. The fibers of the lens at the equator were well preserved and showed well staining nuclei. Centrally, both anteriorly and posteriorly the fibers had broken down to form a fine granular material interspersed with globules.

The zonular fibers were broken here and there and were markedly wrinkled.

Retina and Optic Nerve: Close to the ora serrata the retina became thinner, and the granular layers ran together. At the ora serrata, nasally and above, the retina was hardly recognizable (rupture of the retina at the ora serrata; fig. 8).

In many places close to the pigment epithelium small structureless globules were seen. As these were better seen in the second case, they will be described in the report of that case.

In the macula lutea the granular layers were completely broken, while pigment and pigment cells lay scattered through the outer retinal layers (hole at the macula).

The optic nerve had a deep funnel-shaped cup. This cup was narrow and extended through the lamina cribrosa into the optic nerve. It was impossible to determine how far it extended into the optic nerve, as the latter had been severed close to the globe. The columns of the fibers of the optic nerve anterior to the

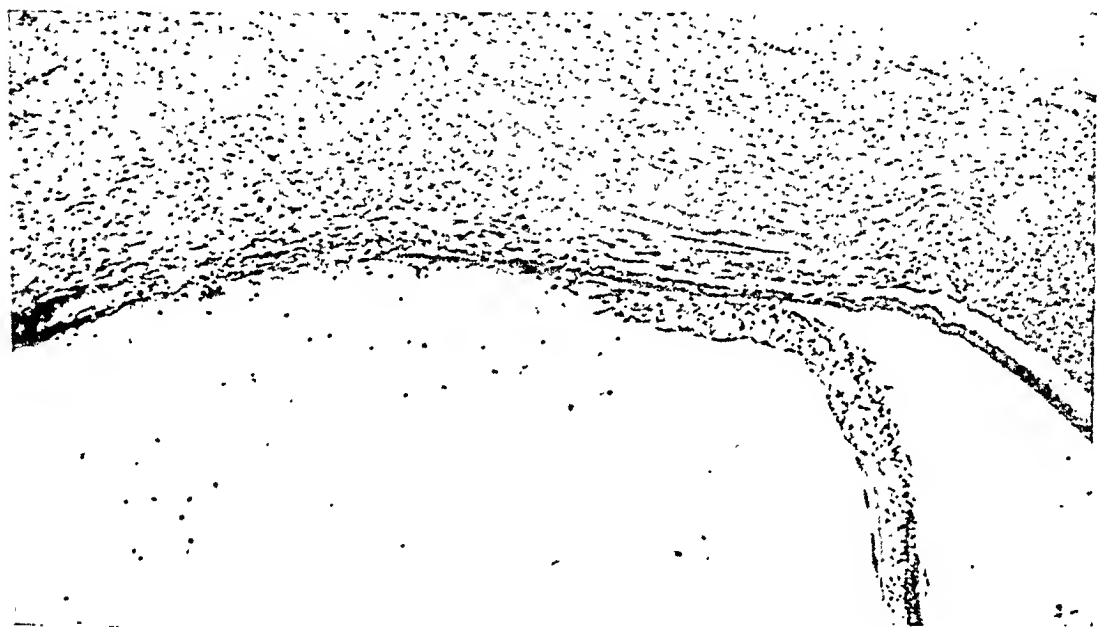


Fig. 8.—The sclera, choroid and retina. The choroid and retina are torn in the same region where the sclera exhibits marked changes in structure and staining. Note the fluid in the epichoroid.

lamina cribrosa appeared to be dislocated backward (rupture of the optic papilla associated with rupture of the lamina cribrosa, Tillema, 1936).

Vitreous Body: The vitreous was closely filled with erythrocytes the contours of which were just visible under high magnification. In addition fine granules staining with eosin and lying close together were observed everywhere. Between these granules many cells were seen to be in varying stages of degeneration. The majority possessed an oval, eccentric nucleus and a finely granular protoplasm. Some had vacuoles, others contained dark brown pigment granules. In a few places, near the ora serrata, the pigment could be demonstrated to contain iron.

CASE 2.—Clinical History.—T. de L., a manual laborer aged 30, received a blow on the left eye with a piece of wood. He was examined the same day (April 13, 1931).

The right eye was normal and emmetropic, with visual acuity of 1.

The left eye showed a fairly large hematoma of both eyelids and the surrounding tissues. The lids were torn in several places. The bulb was hardly seen through the swollen lids and the conjunctival edema. The cornea was hazy and the pupil wide; the iris was perhaps torn. The patient bled from the nose, complained of dizziness and vomited but refused to be admitted to the clinic.

On April 21 he was admitted to the neurologic clinic because of signs of *comotio cerebri*. Nothing serious was found, however, and the next day he was transferred to the ophthalmologic clinic because of the raised tension of the eye.

The cornea was then steamy and the pupil wide and irregular. Behind the pupil the movable, cataractous lens could be seen. The tension was from $+2$ to $+3$.

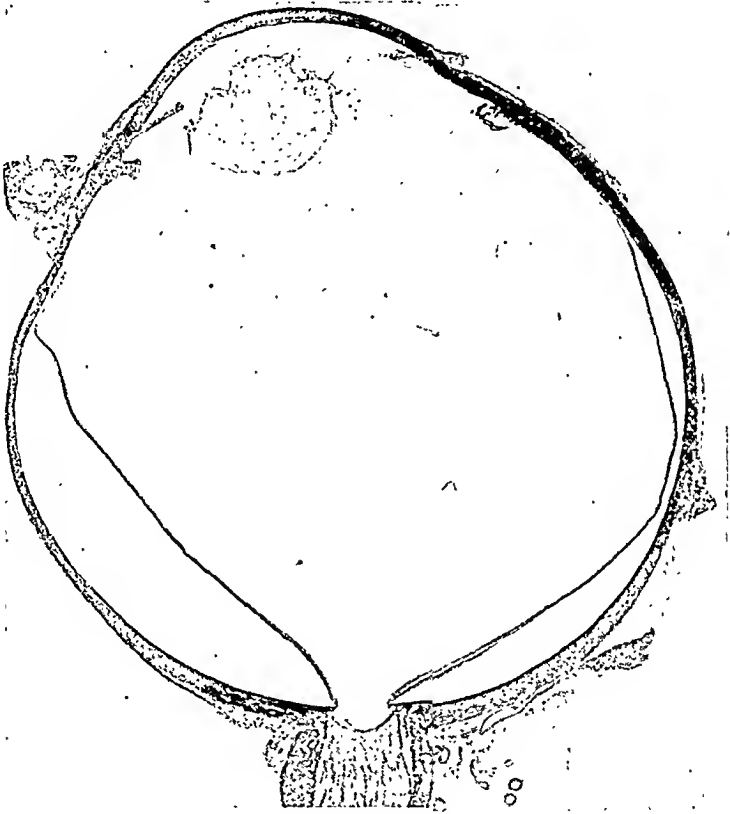


Fig. 9 (case 2).—General view of the eye. The sclera is thinner nasally (left) than temporally. The ciliary body is very thin, and there is a glaucomatous cup. The anterior displacement of the lens is an artefact.

On May 9 the patient left the clinic of his own accord, although he was urged to stay. Some time later he returned. The tension was then $+3$, and the globe had to be removed (June 18).

Microscopic Examination (fig. 9).—Cornea: The epithelial surface was irregular. Here and there the superficial cells appeared to be missing, while in other places they were swollen and vacuolated. The vacuoles had fused and had formed vesicles of varying size. The largest were as big, approximately, as a few cells put together.

Sclera: The sclera had been incompletely ruptured anterior to the equator. This rupture has been described in detail elsewhere (Tillema, 1936).

Vessels in the Sclera: Schlemm's canal had been obliterated in one place medially. It had been replaced by spindle-shaped cells and cells with a large, pale, oval nucleus and by a little pigment.

The anterior ciliary vessels showed pathologic changes in many places. This was well illustrated by an artery running from the internal rectus muscle through the sclera into the ciliary body. In the belly of the muscle the vessel was unimpaired. Near the insertion it was surrounded by closely packed round cells.



Fig. 10.—An anterior ciliary artery penetrating the sclera. The lumen is destroyed, and pigment (both uveal and hematogenous) is scattered in the surrounding tissue. Note the irregular structure of the sclera surrounding the vessel near the ciliary body (below). Above, the artery branching off to the right ends abruptly.

Slightly more anteriorly spindle cells and one or two pigment granules were also observed. The endothelium was swollen, and the wall of the vessel was damaged. Nearer to the sclera these changes became more marked, while at the point of entry (fig. 10) into the sclera the lumen of the vessel was missing. Connective tissue, spindle cells, cells with a large, pale nucleus and small round cells occupied the site of the vessel. In the inner layers of the sclera the same picture was seen, but here a great deal of pigment was scattered about. This pigment was to a

large extent hematogenous. The surrounding sclera had taken the stains poorly and contained various cells and scattered pigment. An anterior branch of this artery had also been obliterated (fig. 10).

One other spot, in the lateral portion, deserves description. Just in front of the anterior ciliary processes the ciliary epithelium had been depigmented and almost interrupted. Large cells containing pigment were lying loose in the posterior chamber and in the ciliary body. If one traced this spot in the serial sections one observed pathologic changes in the inner scleral lamellae similar to those described under partial rupture of the sclera elsewhere (Tillema, 1936). Still farther the remains of a blood vessel were seen, and in tracing this one noted that it was intact in the outer layers of the sclera (rupture of the ciliary body and the inner layers of sclera at the point of entry of a blood vessel).

In the long posterior arteries also pathologic changes were noted. The nasal artery suddenly showed remarkable thinness and poor structure of its wall in the inner layers of the sclera (fig. 11). The temporal artery had no lumen at its point of entry into the choroid, while its endothelium was missing. Its wall had taken the stains badly here and had an irregular structure (fig. 12).

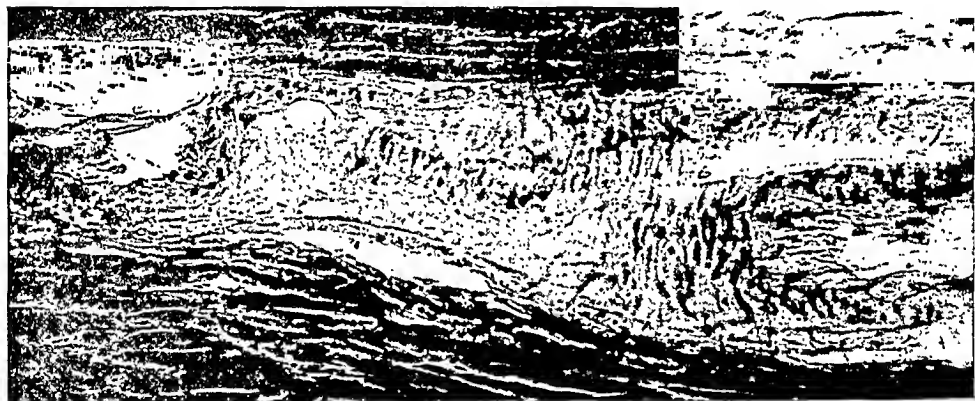


Fig. 11.—A long posterior ciliary artery (nasal artery), showing an exceedingly thin, almost structureless wall in the inner layers of the sclera.

Four vorticosc veins were present. Their perivascular channels were filled with hematogenous pigment. In the inner layers of the sclera, however, this pigment was mixed with pigment derived from the choroid. In one vein the pigment was seen lying in the wall of the vessel against the erythrocytes filling the lumen of the vessel (fig. 13). In another vein pigment was seen lying inside the lumen (fig. 14), while a little farther erythrocytes were seen to fill the spaces surrounding the vein (fig. 15). While the first three veins showed small endothelial defects covered with groups of white blood cells, the fourth vein showed a complete defect of its wall close to the choroid. The surrounding sclera was stained irregularly and contained spindle cells and granules of blood pigment.

The pigment which was seen in the perivascular channels of all these veins could be traced outward into the episclera and from there backward toward the posterior pole of the eye (fig. 16).

Ciliary Nerves: There was a notable contrast between the good structure of most nerves and the partial degeneration of others. The isolated degeneration of one group of myelin sheaths, with good preservation of the rest of the bundle, is demonstrated in figure 17.

Anterior Chamber: The anterior chamber had been completely deformed through the anterior displacement of the iris and lens. The iris, at its periphery, was adherent to the cornea and had closed the angle of the anterior chamber.

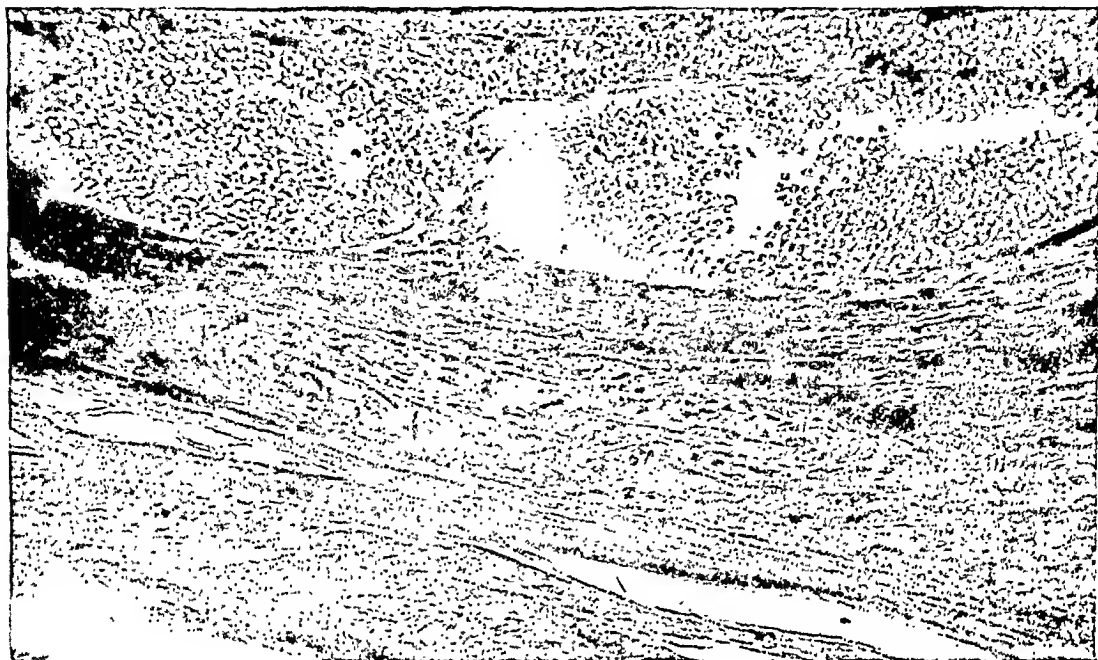


Fig. 12.—A long posterior artery (temporal artery). The lumen is closed; the endothelium is missing, and the wall is irregular in structure (section through the inner layers of the sclera).

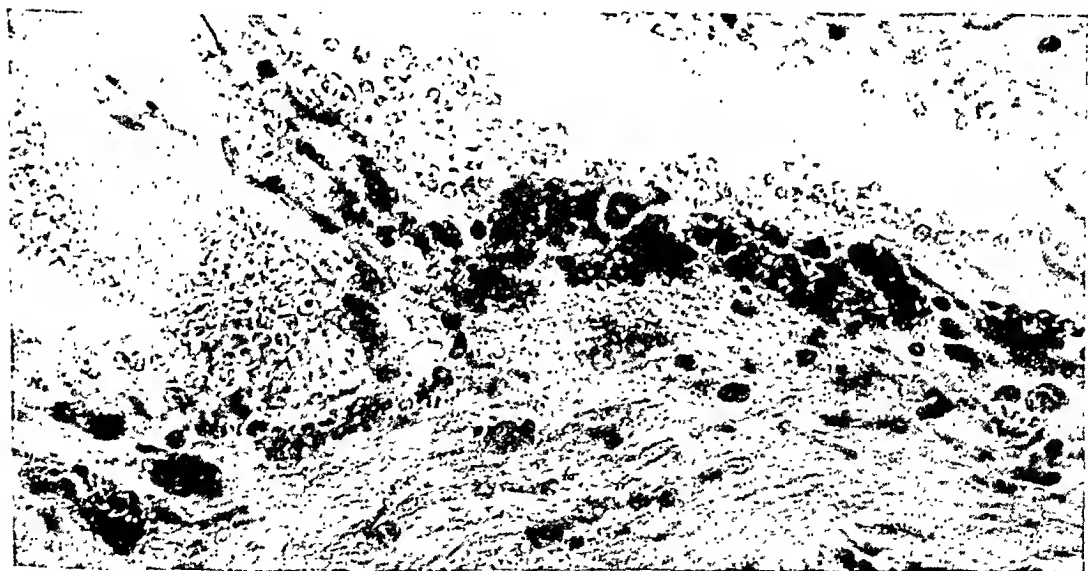


Fig. 13.—A vortex vein, showing erythrocytes inside the lumen and pigment granules in the wall touching each other.

Nasally the vitreous body had entered the anterior chamber and was seen in front of the lens. Continuing on its way in a temporal direction and passing between iris and lens, it had entered the posterior chamber temporarily. The anterior vitreous membrane in this region was complete.

In addition to the vitreous the anterior chamber contained only a few pigment-laden cells.

Iris (fig. 18): The iris was adherent to the cornea peripherally and had closed the angle of the anterior chamber. Its tissue had taken the stains poorly and showed little structure. The nuclei of the cells had a varying depth of color

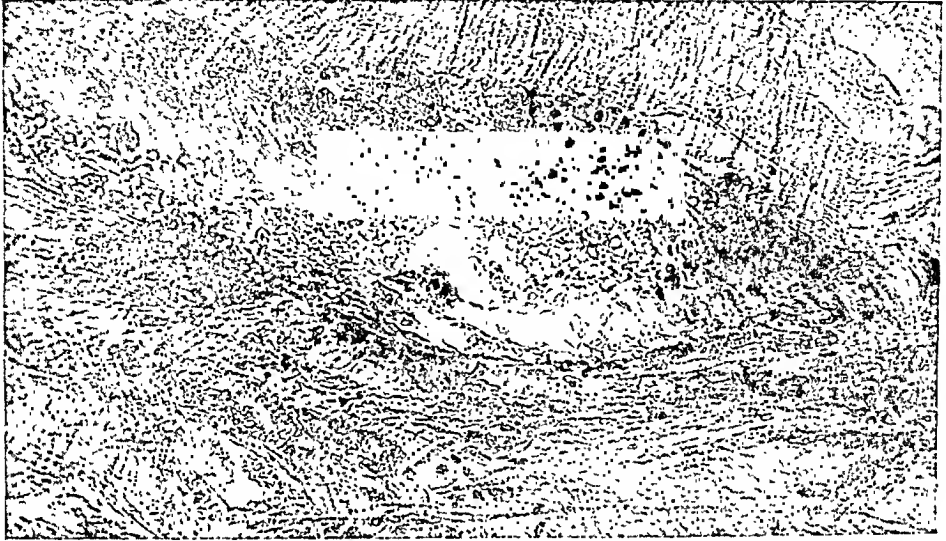


Fig. 14.—A vortex vein, showing pigment granules inside the lumen.



Fig. 15.—The same vortex vein as in figure 14. The lumen of the vein is seen below. Above are seen perivascular channels filled with erythrocytes.

or had shrunk; the walls of the vessels were stained homogeneously with eosin, while the endothelium was swollen or missing. Laterally a large piece of the uveal portion of the iris was missing. In other places medially the whole sphincter portion was missing, while the pigment layer had curled up. In other places tears of the dilator and sphincter muscles and of the stroma were seen.

Ciliary Body: Nasally the epithelial pigment had been scattered into the posterior chamber and into the surrounding tissue of the ciliary body. It was seen as large, round lumps (loose epithelial cells?) in the posterior chamber; in the epithelium and in the surrounding tissue, on the other hand, it appeared as fine, diffusely spread granules. In sections stained for iron no iron was observed here, although it occurred in many places between the ciliary body and the sclera.

Temporally, in a circumscribed area, the anterior ciliary processes contained dense connective tissue with few nuclei and a small amount of scattered pigment.



Fig. 16.—A vortex vein (stained for iron). Above, the vein is seen close to its point of exit from the sclera. Note the closely packed hematogenous pigment around the vein. It may be traced toward the posterior pole (left) of the globe.



Fig. 17.—A ciliary nerve. On the right are well stained myelin sheaths. On the left is a larger bundle in which the myelin sheaths are partly missing.

Just anterior to these processes the epithelium had been destroyed. The ciliary body was very thin here, and bending outward it lined a scleral pit. Traces of iron were noted in this region. Under the section on the ciliary vessels I have described the presence of a damaged vessel in this part of the sclera (rupture of the ciliary body at the point of entry of a ciliary vessel).

The ciliary muscle as a whole was very flat and appeared to contain much connective tissue. In some places pigment granules were grouped around a vessel, generally in the neighborhood of tissue changes.

Choroid: Nasally and above, the choroid and the retina had been destroyed. They had been replaced by connective tissue showing many elongated dark nuclei. This tissue gradually merged into the sclera, which was pale in this region and contained many nuclei (rupture of the choroid).

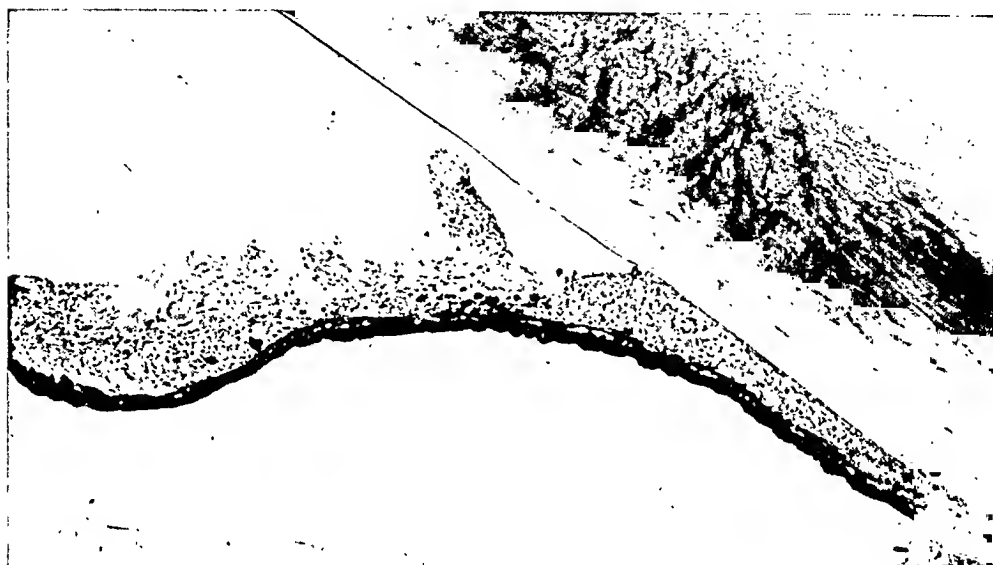


Fig. 18.—The iris, showing necrosis, a tear of the anterior lamella and a thin base adherent to the cornea.

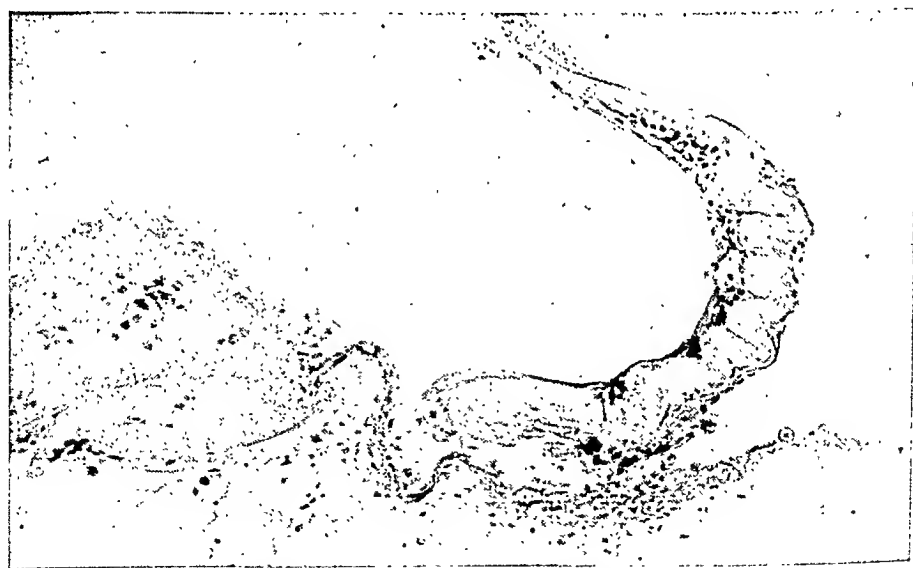


Fig. 19.—The capsule of the lens and the anterior part of the vitreous membrane, both covered with cells derived from the epithelium of the lens.

From the ora serrata backward to a distance far behind the equator the pigment of the epithelium and the chromatophores had been scattered. This was more marked nasally. In a few places the choroid had been completely depig-

mented; it was thin, and consisted of a few elongated cells with spindle-shaped nuclei.

Temporally and behind, smaller ruptures of the choroid were observed. They were represented by groups of closely packed spindle cells which had broken through Bruch's membrane and through the pigment epithelium. Between these cells iron was seen. The outer layers of the choroid had been fairly well preserved. The retina, which had been artificially detached, was adherent to the choroid here.

The outer layers of the choroid and the epichoroid were closely packed with pigment granules. This pigment contained a great deal of iron.

Lens and Zonule: The lens had shrunk. It had been displaced anteriorly (post mortem), and its capsule had been torn. The epithelium had grown around the rent along the hyloid membrane and along the zonule (fig. 19). The normal structure of the lens had been largely destroyed: its substance was granular, and

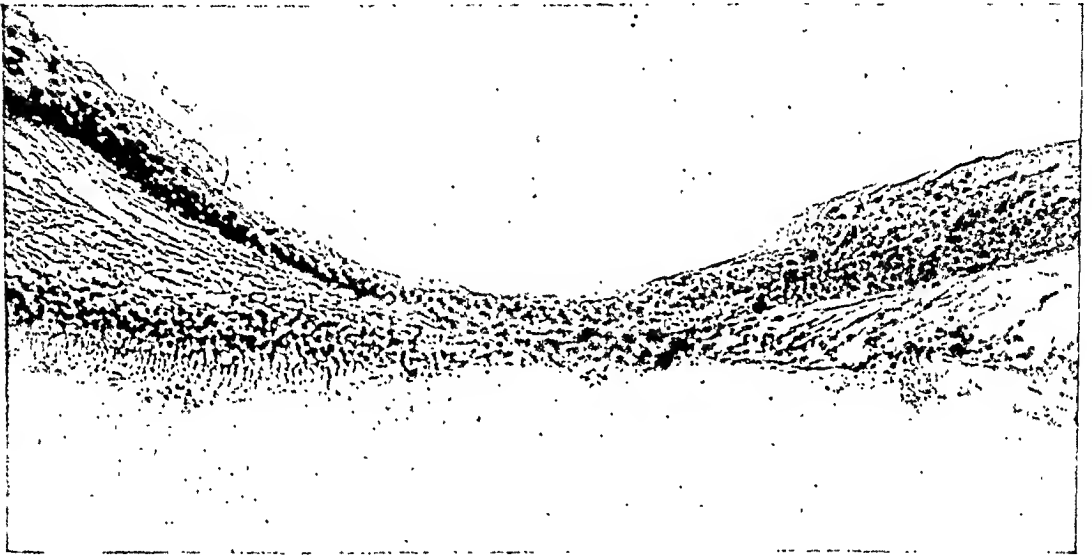


Fig. 20.—The macula lutea. The cones are missing in the fovea. Note the pigment granules in the fovea and the subretinal fluid.

the epithelium was missing in many places. The meridional zonular fibers had been torn nasally, while the radial fibers were missing. Only remnants of the latter were observed temporally.

Retina: The retina was torn in the same place as the choroid. In the macula lutea the cones were missing. Pigment cells and loose pigment were scattered throughout the remaining layers (fig. 20).

The neuro-epithelium was separated from the pigment epithelium by fluid (fig. 21). The neuro-epithelium at the equator had degenerated into small globules of varying size and shape (fig. 21). The majority were round and stained with eosin and with fuchsin. Interspersed between these globules lay large cells with an oval or kidney-shaped pale nucleus. They contained pigment, partly hematogenous but partly also resembling the pigment of the pigment epithelium. Both the globules and the large cells were observed close to the pigment epithelium. The pigment epithelium was swollen. A few cells had even assumed a semi-globular shape (fig. 22) or had formed into a large structureless mass resembling the small loose globules. Just as in the choroid, the changes in the retina were more marked nasally.

Optic Nerve: The optic nerve has been fully described elsewhere (Tillema, 1936).

The optic papilla and the lamina cribrosa had been incompletely ruptured in a backward direction. In this region the central vein had also been torn, and the vitreous protruded into it. In addition, there was a glaucomatous cup.



Fig. 21.—The retina. The rods and cones are missing. Note the subretinal fluid and, close to the pigment epithelium, the small structureless globules.

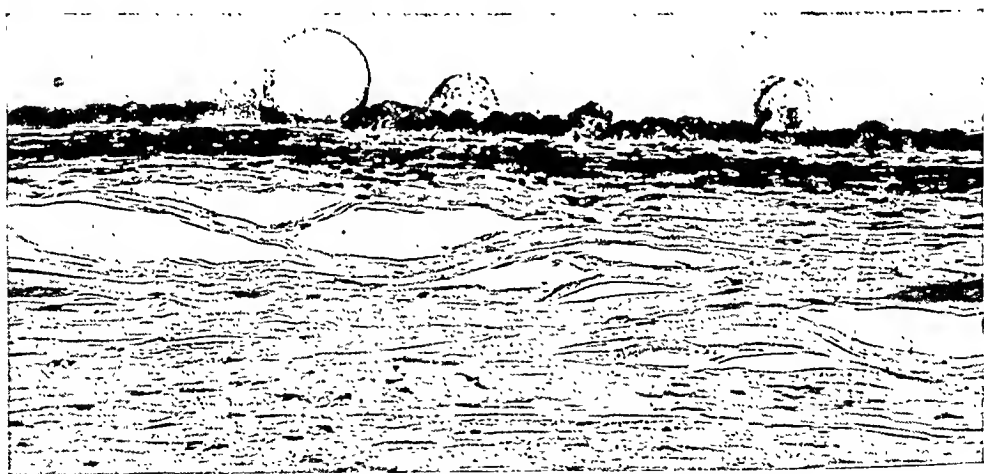


Fig. 22.—Pigment epithelium, showing "inflated" cells.

Vitreous Body: The anterior hyaloid membrane had been torn medially. The vitreous at this point had entered the anterior chamber.

The vitreous contained many scattered cells, mostly with a nucleus varying in shape from oval to segmented. The cells carried many pigment granules that con-

tained no iron. The space between the cells was taken up by a fine granular pink material with small round cells here and there. Many cells showed signs of degeneration to a greater or lesser degree.

Near the equator of the lens and also anterior to the lens in the anterior layers of the vitreous a circumscribed group of red and white blood cells was seen.

COMMENT

Although the two globes just described differed in small details, their resemblance is obvious. No signs of a perforating wound or of infection were noted. The pathologic changes may conveniently be divided into two groups: a large group of destructive changes and a smaller group of regenerative changes. It is obvious that the regenerative changes could not but have been preceded by the destructive changes and that the latter might have been caused partly by the trauma, but that they might also have arisen through unfavorable physical or chemical conditions. As in the majority of cases the changes occurred immediately after contusion (see "Clinical Part"), those changes that were directly caused by the trauma were most likely to give the key to the problem of traumatic glaucoma. For this reason this group of changes will be separated from the others.

All formation of new vessels, all formation of scar tissue and all transport of débris must be regarded as regenerative changes. Formation of new vessels was seen only in the cornea in the first case. Formation of scar tissue was seen in the choroid and sclera. Transport of débris, however, was seen in many places, especially in the vitreous and around the vessels in the sclera. The numerous loose pigment granules were partly hematogenous and partly derived from the uvea. Although both kinds usually occurred together, it must be noted that the pigment around the vorticosae veins was almost exclusively hematogenous. It was interesting to see the growth of lenticular epithelium (my second case) along the torn capsule and the anterior part of the hyaloid membrane. This illustrates that the lens too is a living tissue.

The remaining changes were destructive. As some of them may be observed in eyes that have never suffered a trauma, it seems unlikely that they should constitute a primary factor in the origin of traumatic glaucoma. For this reason these also must be separated and laid aside.

The changes which have been observed in the corneal epithelium and stroma are familiar. They have often been described as regards both their clinical aspects and their microscopic aspects in cases of glaucoma. The changes in the sclera have been fully discussed in a separate article and constitute indirect, incomplete ruptures. Adherence of the peripheral part of the iris anteriorly, thinness of its basis and infiltration by pigment of the trabeculae of the pectinate ligament have often been observed preceding intercalary staphyloma in cases

of primary glaucoma. Moreover, according to the clinical history in my cases, the adherence of the peripheral part of the iris did not exist at first. This, then, must be regarded as a secondary change, a view supported by Garnier's observation that in his case the angle of the anterior chamber was normal.

Necrosis of the iris is seen in association with many different conditions. I know from personal observation that it may occur in advanced stages of glioma of the retina with secondary glaucoma, in cases of choroidal sarcoma and in cases of leprosy. It has been described (A. Fuchs) as occurring in cases of *ulcus serpens corneae*, in cases of traumatic ring abscess of the cornea associated with endophthalmos, in cases of necrotic sarcoma of the choroid and in cases of glaucoma. Necrosis of the iris, therefore, may be a secondary change, but it has been described after contusion (A. Fuchs). Moreover, the hematogenous pigment scattered irregularly throughout the stroma of the iris suggests a traumatic lesion. Another reason for thinking this is the fact that the ciliary body exhibited similar changes in a circumscribed area (my first case), while in glaucoma the changes would be more general. If the pigment granules in the iris were grouped around the vessels one might be tempted to think that the vessels had formed drainage channels for the pigment, such as Gerbrandy hypothesized from his investigation or such as Rochat observed in his experiments with hemoglobin. There is no such grouping, however, and therefore the pigment granules must be remnants of hemorrhage. Finally, I have observed necrosis of the iris in a case of contusion caused by an exploding cartridge. One may conclude, therefore, that at least part of the necrosis of the iris had been caused by contusion.

In my first case both the anterior chamber and the posterior chamber had been deformed by displacement of the iris and lens. The wrinkling of the capsule of the lens suggests a slackening of the zonule *in vivo*, a likely view if one remembers that the zonular fibers are partly torn. Probably, therefore, the lens was slightly dislocated *in vivo*. In my second case the anterior forward displacement of the lens must have been an artefact.

The circumscribed necrosis of the ciliary body (my first case) has already been mentioned. The same arguments brought forward for the iris apply here. The thinness of the ciliary body and its elongation in a backward direction may have been caused partly by distention of the sclera but partly also by atrophy such as is seen in glaucoma.

Detachment of the choroid and of the ciliary body is also observed after intra-ocular operations and generally in hypotony of the globe. It is remarkable, therefore, that it occurred in both my cases and in Garnier's case of traumatic glaucoma. It may be explained by disturbance of the intra-ocular circulation.

The cupping of the optic papilla (my second case) is similar to the cupping in glaucoma, but in addition both my patients showed a tear of the lamina cribrosa and the optic nerve. The latter must have been caused by contusion (Tillema). Structureless globules between the pigment epithelium of the retina and swelling of the epithelial cells such as I have seen have been described by Koyanagi in cases of nephritic retinitis and eclampsia. From the observation of intermediate stages between the cells and the globules Koyanagi concluded that in his cases the globules had been actively secreted by the epithelium. As I did not observe such intermediate stages, the globules in my cases must be regarded as a form of degeneration based on circulatory disturbance in the choroid such as was described by Krückmann in 1899.

Thus one arrives at the last group of changes, i. e., those caused directly by contusion. In both cases this group consists of rupture of the inner layers of the sclera inward and upward, rupture of the lamina cribrosa, destruction of Schlemm's canal medially, lesions of the ciliary vessels and nerves, endothelial changes in the vortex veins, tears of the iris and (partly) its necrosis, circumscribed necrosis of the ciliary body and rupture of the retina and choroid. In addition, my second patient showed rupture of the anterior capsule of the lens and of the anterior hyaloid membrane, with prolapse of vitreous into the anterior chamber, and, finally, rupture of the central vein at the optic papilla. These differences between the two cases are important.

Although it is well known that both rupture of the capsule of the lens and thrombosis of the central vein may be followed by glaucoma, these must be exceptional causes of traumatic glaucoma, as neither rupture of the capsule of the lens nor rupture of the central vein was present in one of the cases collected from the literature and in one of my clinical cases (See "Clinical Part").

Some of the changes common to both cases also require further discussion. The lesions of the vessels in the sclera were so unusual that they could have been caused only by trauma. Nevertheless, changes in the vortex veins have also been observed in other forms of glaucoma. Hypertrophy of the endothelium obliterating the lumen of the vessels and obliteration of the perivascular channels through hypertrophy of tissue with few nuclei have been described, but these changes are not constantly present (Stirling, Köllner and Thomsen). According to Elschnig, they are more frequent and more marked in cases in which the condition is of long standing, and it has not been proved that these changes cause glaucoma. All these changes, however, are entirely different from what I observed. Partial closure of the veins without anatomic changes such as was described by Schieck (1934) in a case of malignant glaucoma is also different.

It is possible, however, that in traumatic glaucoma the veins may be closed by thrombi caused by a traumatic lesion. It has to be decided, therefore, whether the material observed in some of the veins represents thrombi.

It will be remembered that a lesion in the wall of a vessel must be accompanied by circulatory disturbance to cause formation of a thrombus (Dietrich, 1932). Possibly these two factors were present, but even so the absence of organization which is generally easily recognized after even a few days (Dietrich) renders a thrombus unlikely. Even if organization could be accepted in a few places this organization was so little marked that the thrombi must have been very young and could not have played a part in raising the intra-ocular tension. It is almost certain that they represented postmortem clots.

The granules of hematogenous pigment scattered around the anterior ciliary vessels and the vortex vessels must have been remnants of previous hemorrhage. They confirm the previously expressed opinion that the vascular lesions were traumatic. These granules were also observed in the iris and ciliary body, and they appeared to have been transported from the inner part of the eye along the epichoroidal and perivascular channels of the vorticoso veins into the episclera. Thence they could be traced back toward the posterior pole of the globe.

Degeneration of the ciliary nerves also occurs in nontraumatic glaucoma. Isolated degeneration of a few bundles, however, in a nerve the other bundles of which have been well preserved is a striking phenomenon. There is no reason why this should occur in other forms of glaucoma, and it is suggestive of a traumatic lesion.

In summary, one may say that the following anatomic changes are probably directly related to the accident: partial rupture of the sclera, pectinate ligament, lamina cribrosa and optic nerve; tear and partial necrosis of the iris; necrosis of the ciliary body; rupture of the choroid and retina; rupture of ciliary and vortex vessels in the inner layers of the sclera (probably related to rupture of the sclera); subluxation of the lens (my first case), and isolated degeneration of bundles of the ciliary nerves.

The hemorrhage into the vitreous had been caused by vascular lesions and might have occurred immediately or later. The same applies, of course, to other hemorrhages.

Having analyzed the results of the anatomic study, one is tempted to propound a theory on the origin of traumatic glaucoma. The reader will have noticed, however, that even in the present discussion it has been necessary to refer to data that have issued from clinical study.

It is better, therefore, to await the result of clinical study and to discuss the anatomic data with the clinical data.

II. CLINICAL PART

REPORT OF FIVE CASES OF TRAUMATIC GLAUCOMA

After examining the microscopic sections one is, of course, interested to know whether similar changes can be seen in the living subject. I have been fortunate in having had the opportunity of examining a number of patients with traumatic glaucoma, although this condition is not often seen. The cases have been arranged according to the severity of the attack.

CASE 3.—T. J. S., a fitter aged 45 years, was hammering a brass tube when a piece of brass struck his eye with considerable force. He was examined the same day (March 18, 1933).

The right eye was normal, and visual acuity was 1. Refraction showed hypermetropia of 0.5 D.

The superior lid of the left eye was swollen, and the bulbar conjunctiva was torn medially and laterally. There was a deep wound of the cornea laterally and various erosions. Descemet's membrane was wrinkled. The iris was torn medially. The pupil was wide, and blood coagulum was seen adhering to the iris. The lens was clear on transillumination. No gross changes were seen in the fundus. Sideroscopic examination gave negative results.

On March 20 the lens was seen to be mobile; there was blood, but no exudate, in the anterior chamber.

On March 24 the lens turned diffusely turbid and began to move backward medially. The tension was slightly too high ($+\frac{1}{2}$).

On March 31 the tension was from $+\frac{1}{2}$ to $+1$.

Notwithstanding the administration of pilocarpine the tension rose higher. The patient was then admitted to the clinic, and the lens was extracted. Recovery was uneventful.

The vitreous cleared gradually. The patient was last seen on July 31, 1934. The cornea was then clear, and the anterior chamber deep. The pupil was wide, and it did not react to light or in convergence. A large artificial coloboma of the iris was present medially and superiorly. The stroma of the iris was very atrophic (both the anterior and the posterior lamella). The light reflected from the retina was seen through fissures in the stroma of the iris. The vitreous body was clear except for a few opacities. The fundus was well seen through the wideness of the pupil. The optic papilla was normal. The macula showed irregular pigmentation occupying an area slightly larger than the macula. The retina and choroid were normal except in the superior nasal quadrant, where the pigment of the ora serrata was scattered on a gray background showing a few white spots. A short distance behind the ora serrata also a small amount of pigmentation was seen. This pigmentation was coarse and was probably choroidal. The ciliary processes had been partly depigmented; they were seen as grayish-white masses alternating with darker masses. Part of the pars plana of the ciliary body was also seen.

The ocular tension was normal, as was the projection of light. Visual acuity was 1/300 and was but slightly improved by glasses. The field of vision revealed a large central scotoma.

In this case the ocular tension rose on the sixth day after contusion. The slight medial dislocation of the lens was probably only a coincidence. The rupture of the zonule, the prolapse of vitreous into the

anterior chamber, the medial tear of the iris, the hemorrhage into the vitreous and the pathologic changes of the retina and choroid indicate that this eye suffered a serious lesion. As many of the lesions were situated medially and superiorly, they must have been related. If one remembers that the majority of ruptures of the sclera are also situated superiorly and medially and if one recollects the description of the microscopic features in my first two cases, one is struck by their similarity. In every case pathologic changes were most marked superiorly and medially. In every case the changes in the retina and choroid consisted of loss of structure, pigmentation and slight formation of scar tissue. Changes were also seen in the ciliary body. One may be sure, therefore, that the clinical and the microscopic pictures correspond and that the clinical picture may be interpreted as representing a tear of both the retina and the choroid and also perhaps of the inner layers of the sclera along the ora serrata.

In addition, there was in this case a traumatic lesion of the macula such as was described almost simultaneously by Haab, Kuhnt and Ogilvie in 1900. The anatomic substrate of this lesion has been described in the report of the first two of my cases (first section).

It must be noted that in this case the tension remained high notwithstanding the fact that the blood was being resorbed. The tension dropped to a normal level after the removal of the lens. This fact cannot be used to support the theory that the lens caused the high tension, as the beneficial effect of the extraction of the lens might just as well be interpreted in favor of opening the anterior chamber. The rôle of the lens in the causation of traumatic glaucoma will be discussed later.

In the iris loss of structure of the anterior lamella, depigmentation of the posterior lamella and thinness of the whole iris were found. These were also observed on microscopic examination in both my cases reported in the first section. Tears of the iris were also noted in every case. The condition of the iris, therefore, must have been similar in all three cases.

CASE 4.—J. C. S., a manual laborer aged 34, was struck in the right eye on Aug. 21, 1934, by a piece of wire hanging from a passing motor van. Immediately his eyesight was hazy. The next day he had severe pain. His physician found that the anterior chamber contained blood and administered a drop of atropine. Three days later the patient could still count fingers at a distance of about 15 feet (4.6 meters). That night he had severe pain, and in the morning he found that he could not see.

He was examined in the Eye Clinic of the University of Utrecht on August 25. Visual acuity was found to be perception of light only; there was total hyphemia. No perforating wound was seen. The tension was $+1\frac{1}{2}$. The left eye was normal. Visual acuity was 1, with hypermetropia of 0.75 D. The patient was advised to go to the hospital in his home town.

That same day he was admitted to the University Eye Clinic in Amsterdam. It was found that the eyelids and the bulbar conjunctiva were slightly swollen. There was not much ciliary injection, and no wounds were seen. The cornea was smooth (no vesiculation of epithelium was present). The anterior chamber was completely filled with dark blood. The globe was sensitive to touch. The tension was very high, +3. Vision amounted to perception of light only. The left eye was normal. The tension, according to Schiötz, was 75 mm. in the right eye and 20 mm. in the left eye.

Pilocarpine was prescribed.

On the following day the tension was the same. The anterior chamber was then opened inferiorly. A small amount of fluid escaped, but no blood. The pain was not relieved, and the tension remained high. On the same day, therefore, the anterior chamber was opened once more, this time superiorly. The coagulum was washed out with a fine cannula, and a broad iridectomy was performed. Recovery was uneventful.

The patient was dismissed on September 14. The tension was normal. The fundus was seen hazily through remains of hemorrhage into the vitreous. Visual acuity was counting five fingers at 0.5 meter. There was good projection of light. Examination of the visual field showed that the superior quadrants were missing, probably owing to nests of hemorrhage in the vitreous.

On November 14 the patient was seen for the last time. The cornea exhibited a rest of *Durchblutung*. The iris was markedly atrophic; there was a total artificial coloboma superiorly, two anterior synechia inferiorly, a posterior synechia laterally and remains of exudate of the anterior surface of the lens. There were no dislocation and no trembling. The vitreous showed a few coarse and punctate opacities. The optic papilla was normal. The macula exhibited a sharply defined, round, clear area, surrounded by a dull area. Peripherally, pigmentation was seen along the ora serrata, excepting laterally and inferiorly. This pigmentation was fairly coarse and must have been choroidal. Moreover, the pigment was arranged here and there, especially superiorly, along the choroidal vessels, and in one place it was crossed by a retinal vessel. Here and there the pigmentation was relieved by a paler area. Visual acuity was 1/2 after correction with a -1 cylinder. The visual field was normal.

In the sound eye visual acuity was 1.

In this case glaucoma probably commenced on the fourth day. The patient's complaints before that time may be explained by the contusion and by the hyphemia. On the fourth day, however, he had severe pain, and his visual acuity diminished to perception of light, probably owing to a fresh, large hemorrhage. It is difficult to judge the effect of atropine administered on the second day, because in several cases mentioned in the literature this treatment has been carried out without poor results. However, the effect of mydriatics will be discussed later.

Although the tension was high, the corneal surface remained smooth. The peculiar composition of the blood in the anterior chamber is also remarkable. This must be different from the hyphemia that easily escapes with paracentesis after an operation for cataract. This blood cake probably consisted of a mixture of blood and albuminous exudate such as was mentioned by Morax (1921, eighteenth case) or a gelatinous exudate such as was mentioned by other authors, e. g., Schmidt-Rimpler

(1883), Sala (1904, first case) and Cordes and Horner (1933, first and third cases). In my case to be reported next, also, a glutinous fluid was noticed during the operation, while Stoewer (1904) described a blackish-red mass similar to the mass in my case. Wagenmann expressed the opinion that a gelatinous transudate is not uncommon after serious contusion and that it disappears in a short time. In any case it is important to note that no aqueous escaped when the anterior chamber was opened. This is worth remembering.

The iris exhibited an anterior synechia peripherally and marked atrophy of its stroma; both changes were observed also in the cases in which microscopic examination was made. One is also struck by the extensive pigmentation along the ora serrata, probably caused by a choroidal lesion. The lesion of the macula also must have been traumatic.

CASE 5.—C. G., a grocer aged 35, was struck in the right eye by a belt on May 20, 1935. Although his eye was painful, he could see well for two days. During the night he felt a severe pain and sought his physician's advice.

On May 23 there was marked injection of the pericorneal conjunctiva. The cornea was hazy in its middle part; little light was reflected from the retina, and the latter could not be seen. The tension was $+1$. A bandage was applied.

The tension dropped, but on May 27 the patient had severe pain and vomited. Physostigmine was prescribed, but the tension remained high, i. e., 60 mm. according to the Schiötz scale. Iridectomy was then performed. It was noticed that only a small amount of sticky fluid escaped but that the anterior chamber remained deep. Recovery was uneventful.

On November 26, apart from the total artificial coloboma of the iris, a few opacities were present in the vitreous and a few rests of hemorrhage were observed in its lower part. In the periphery of the fundus, above and laterally and just posterior to the ora serrata the retina was slightly hazy over a small area. This area was surrounded by a small amount of pigment and contained a small horse-shoe-shaped tear with a prominent border. No other changes were found, notably, no dislocation or partial dislocation of the lens. The tension was normal. Visual acuity was normal with glasses. The other eye was normal in every respect, being emmetropic and having normal visual acuity.

In this case a fairly young man had an attack of acute glaucoma on the third day after contusion. It was necessary to carry out iridectomy. It is important to note that the iris in this case was normal apart from the artificial coloboma and also that a hemorrhage into the vitreous had occurred.

CASE 6.—A man aged 20 years was struck in the right eye by a cricket-ball on Feb. 1, 1930. He was examined a few hours afterward. The cornea was then clear. There was a slight hyphemia. The pupil was perhaps a little wider than that of the other eye but reacted well to light. Visual acuity was normal in both eyes.

The hyphemia was resorbed in a few days.

On February 6 the patient returned and said that he was seeing colored rings around the lights. The cornea was steamy, the corneal epithelium vesiculated, and the pupil did not react well to light and was abnormally wide. The tension was from $+2$ to $+3$. Pilocarpine was prescribed.

The following day the tension was normal and the pupil narrow. The patient was kept under observation for a few days. He then went abroad and was seen no more.

This patient suffered a typical attack of acute glaucoma six days after the accident. It should be noted that the attack occurred after the intra-ocular hemorrhage had been resorbed. This patient responded well to pilocarpine.

CASE 7.—G. B., a rammer aged 54, was struck in the right eye by a large piece of wood on Oct. 25, 1934. His eye was painful immediately; he could see no more and felt sick.

Two hours after the accident he was examined. There were slight abrasions of the eyelids. The conjunctiva was intact. The cornea was steamy, and the

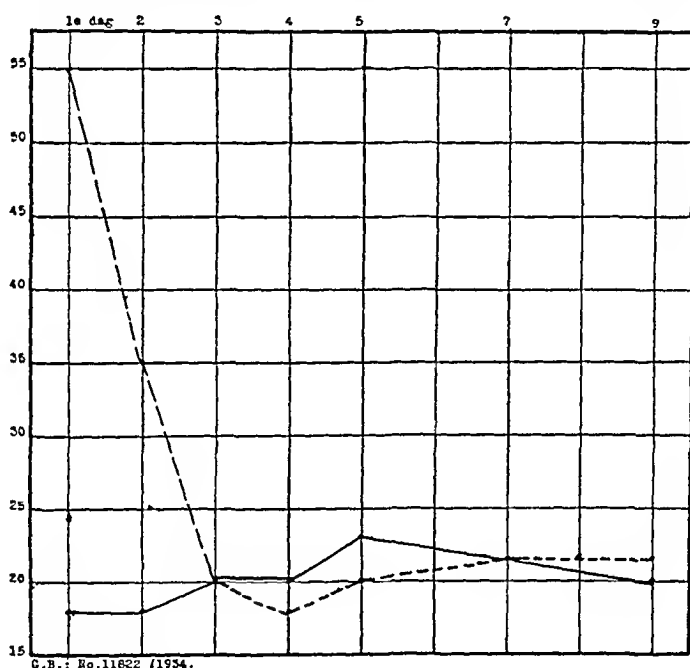


Fig. 23 (case 7).—Diagram showing tension in each eye in the course of traumatic glaucoma of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

pupil fairly wide. No hyphemia and no tears of the iris were present. The fundus was not to be seen. The movements of both eyes were normal. The left eye was normal.

Visual acuity in the right eye was $1/2$; there was no correction with glasses.

Visual acuity in the left eye was 1; the eye was emmetropic.

The tension in the right eye, according to the Schiötz scale, was 55; that in the left (normal) eye was 18. Pilocarpine was prescribed. Figure 23 shows the course of the tension of each eye.

The patient was discharged on November 25. The pupil of the right eye was still somewhat wider than that of the other eye and was slightly irregular in shape. There was slight torsion of the fibers of the iris, but the pupillary reaction to light was equal on both sides. Close examination of the fundus revealed no pathologic changes. The tension was normal; visual acuity was normal, and the eye was emmetropic.

In this case glaucoma, associated with a hazy cornea and wide pupil, immediately followed the trauma. This case is remarkable for the absence of any visible traumatic lesions.

The ocular tension was measured with the Schiötz tonometer during nine days. A glance at figure 23 will show that glaucoma was followed by a longer period of hypotony. Also I wish to draw attention to the similarity between this figure and the following figures illustrating the effects of contusion of the eyeball in which only hypertony but no other sign of glaucoma could be found.

CASES OF SIMPLE CONTUSION

It was found (Leplat and Schmidt and de Decker) that in animals contusion is followed by a short period of hypertony (from thirty to forty minutes) and this is followed by a longer period of hypotony (from three to seven days). As it is impossible to take tonometer

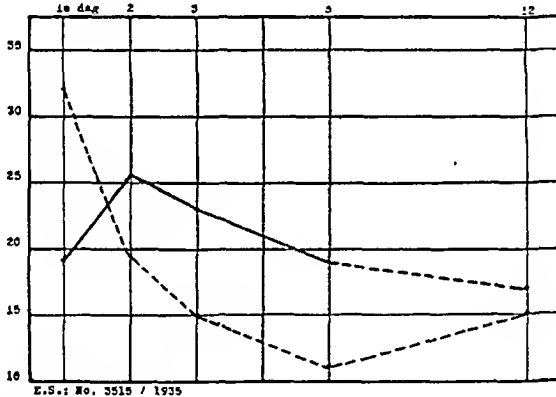


Fig. 24 (case 8).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

readings every few minutes on the human subject, one has to be content with daily readings. Even then one is struck by a certain similarity between the curves for animals (compare the original articles, or Schieck and Brückner's "Kurzes Handbuch der Ophthalmologie," volume 4, page 454), the case of traumatic glaucoma just reported and the following cases of simple contusion.

CASE 8.—E. S., aged 20, a draughtsman, struck himself in the right eye with the metal end of a tape measure on March 14, 1935. He was examined a few hours later.

The right eye showed superficial erosions of the cornea. The pupil was oval in a superomedial direction and wider than that of the other eye. A fairly large hyphema was present; the fundus was not to be seen. Visual acuity amounted to perception of movements of the hands.

The left eye was normal, with normal visual acuity.

Refraction showed hypermetropia of 0.5 D. in each eye.

The tension (Schiötz) was 32 mm. in the right eye and 19 mm. in the left (the sound eye).

The following day the cornea was slightly hazy, and its posterior surface was covered with a fine precipitate. The hyphemia had disappeared; the pupil was still oval, and the optic papilla could be fairly well seen. Visual acuity was 1/6. The cornea cleared rapidly, and the reaction of the pupil returned in a few days. On March 18 visual acuity was normal. However, slight paresis of accommodation remained. On March 19 careful examination of the fundus revealed a circumscribed detachment of the retina superiorly and medially, with several small holes.

On April 2 the detachment of the retina had healed spontaneously. On April 9 the patient said that he had no more trouble with reading at a short distance.

CASE 9.—J. A., aged 15, was struck in the right eye with a metal buckle on March 13, 1935. He was examined a few hours later.

The right eye was normal.

Examination of the left eye showed that the eyelids were slightly swollen. The ocular movements were unimpaired. There was a slight pericorneal injection.

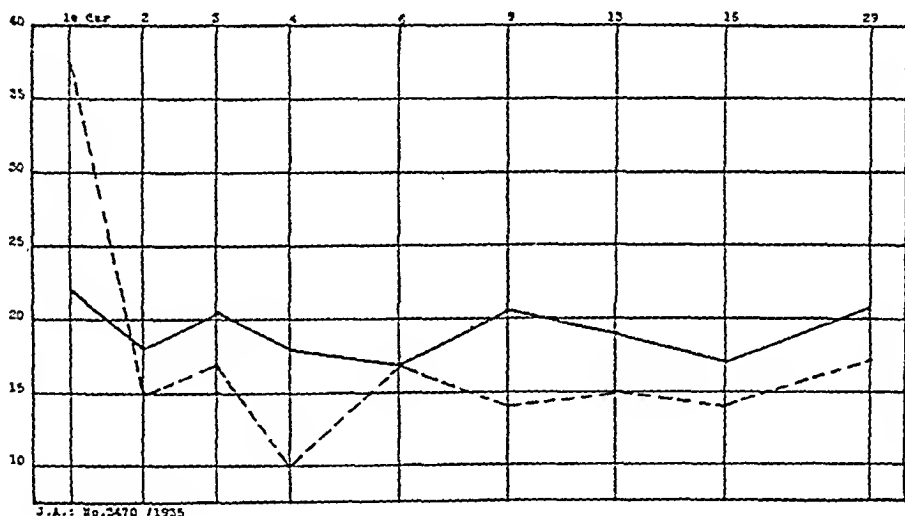


Fig. 25 (case 9).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

The cornea was slightly hazy, smooth and not swollen. A large hyphemia was present in about one third of the anterior chamber; the aqueous was slightly turbid. Slight iridodialysis was present medially and superiorly; the pupil of the injured eye was flattened and wider than that of the other eye. There was no reaction of the pupil to light, and no light was reflected from the retina. Visual acuity amounted to perception of movements of the hand.

The tension (Schiötz) was 22 mm. in the right eye (the sound eye) and 37.5 mm. in the left.

Figure 25 shows the course of the tension.

Pilocarpine was prescribed.

On the following day the pupil was narrow; there was a small hyphemia, and the optic papilla was visible. Visual acuity was normal in each eye. Refraction showed hypermetropia of 1 D. in the left eye and of 1.5 D. in the right.

On April 3 the fundus was examined under the influence of a mydriatic. No pathologic changes were noted.

CASE 10.—J. R., a fitter, aged 24, received a blow with a fist on the left eye on April 11, 1935. He was examined a few hours later.

The left eye showed a small tear of the inferior lid and a small subconjunctival hemorrhage. The cornea was clear. The pupil was slightly oval but was equal in size to the other pupil and reacted as well to light. The optic papilla could be well seen and showed no abnormality.

Visual acuity was normal in each eye; refraction showed hypermetropia of 0.75 D. in the right eye and of 0.25 D. in the left. The fundus was normal.

The tension of each eye may be read from figure 26.

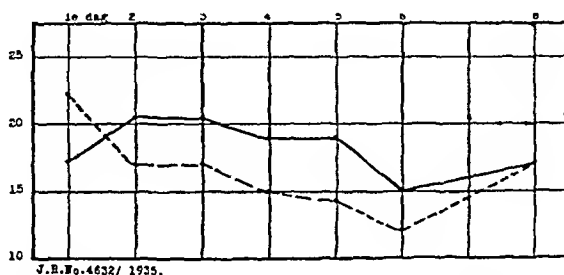


Fig. 26 (case 10).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

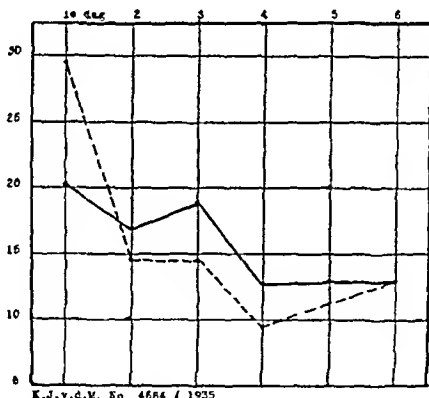


Fig. 27 (case 11).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

CASE 11.—K. J. v. d. M., the driver of a car, aged 28, was struck in the left eye by a piece of wood on April 13, 1935. He was examined a few hours later.

The right eye was normal.

The left eye showed a hematoma of the superior lid; the aqueous was turbid, and there was a small blood coagulum in the anterior chamber. The fundus, as far as could be ascertained, was normal.

Two days later visual acuity was normal in each eye. There was a slight degree of hypermetropia (1 D.). The tension of each eye may be read from figure 27.

After six days the pupil was still slightly oval, but it reacted well to light. No pathologic changes could be observed in the fundus.

CASE 12.—M. L., aged 7, was struck on the right eye by a tennis ball on April 26, 1935. He was examined one and a half hours later.

Examination of the right eye showed a slight pericorneal injection. The aqueous was turbid, with a fine keratic precipitate. The pupil, larger than that of the left eye, was slightly oval in a vertical direction and showed a slow reaction to light.

Visual acuity was $1/6$ in the right eye and normal in the left; both eyes were emmetropic.

The ocular tension of each eye may be read from figure 28. It must be remembered, however, that the first two readings have only relative value, as the child was frightened and could not be examined well. The figures represent the reading found a few times in succession. Later the patient was calm when he allowed himself to be examined. The course of the ocular tension may be read from figure 28.

On the following day the slight keratic precipitate was still present; the pupil was oval in a horizontal direction, and the pupillary border was slightly damaged on the medial side. The anterior chamber was deeper than that of the sound eye, and the iris trembled, i. e., the lens had been slightly dislocated. The fundus was well seen and showed no pathologic changes.

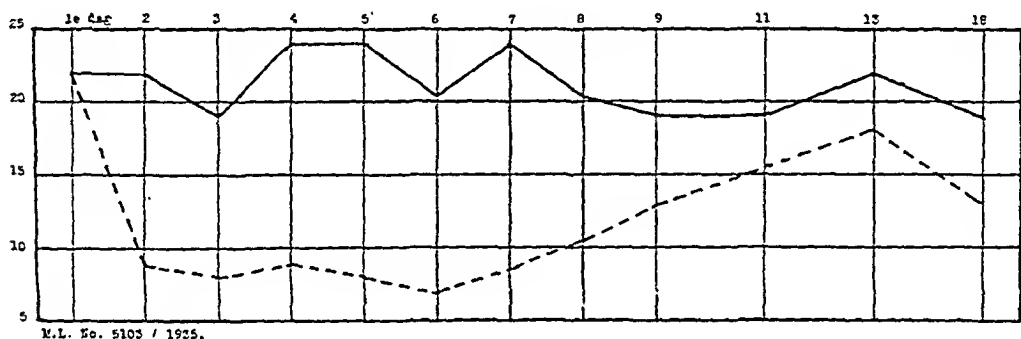


Fig. 28 (case 12).—Diagram showing tension in each eye following simple contusion of the right eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

After eighteen days the pupillary reaction to light returned. Visual acuity was normal.

After this the patient stayed away, so that the fundus was never examined under the influence of a mydriatic. It is possible, therefore, that minor details have been overlooked.

CASE 13.—Nora van V., a girl aged 8, was struck in the left eye by a toy arrow. She was examined within an hour after the accident.

The right eye was normal.

Examination of the left eye showed a small wound of the bulbar conjunctiva medially, with a small subconjunctival hemorrhage. The cornea was a trifle hazy, especially below; its surface, however, was smooth. The iris was greenish. The pupil was round and equal in size to the other pupil but did not react to light.

The child allowed the ocular tension to be measured without any reluctance. The tension was equal on both sides—18 mm. (Schiotz). Figure 29 shows the course of the ocular tension.

Visual acuity was $1/6$ in the right eye (the sound eye); with a cylinder of $+2.5$ D. it amounted to $1/2$.

Visual acuity was 1/6 in the left eye, with a cylinder of +3 D. it was 1/2 partly.

This low visual acuity is explained by the fact that this child, although suffering from astigmatism, had never worn spectacles.

After eight days the pupillary reaction to light was normal again.

On May 21 the fundus was examined under the influence of a mydriatic to get a better view of the picture that had been noted before. Medially and inferiorly a sector-shaped pale area was seen, with the broad part of the sector turned to the ora serrata. This area was paler than the surrounding retina and showed scattered fine dots and somewhat coarser pigment dots. The fine dots seemed to be arranged along the choroidal vessels in a few places. The coarser dots, of which only a few were seen, were surrounded by a small yellowish-white ring, and their size corresponded to the size of the choroidal vessels. In one place three of these dots were seen lying in a row in front of a choroidal vessel. One of the choroidal vessels was flanked by yellowish-white stripes.

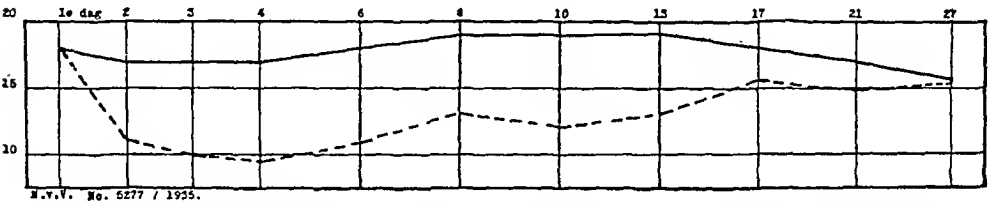


Fig. 29 (case 13).—Diagram showing tension in each eye following simple contusion of the left eye. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

TABLE 1.—Tension in Millimeters for Case 14

Date	Right Eye	Left Eye	Difference
10/24/35.....	22	17	+5
10/25/35.....	18	18	0
10/28/35.....	17	17	0

In the following cases the patients were not examined so often. Each one, however, was seen on the day of the accident. Generally an insignificant contusion of the globe caused the patient to seek advice.

CASE 14.—J. C. B., aged 30, was struck in the right eye by a jet of water. He was examined the next day.

Visual acuity was normal for each eye, with hypermetropia of 1.5 D.

Examination of the right eye showed that the pupil was smaller than that of the sound eye. The reaction to light was good. Otherwise, the eye was normal.

The tension according to Schiötz is shown in table 1.

CASE 15.—J. B., aged 59, was struck in the right eye by a piece of wood. He was examined the same day.

The right eye was emmetropic. The results of tests of visual acuity were not reliable, and there was a large hyphemia.

The left eye had normal visual acuity with a cylinder of +0.5 D.

The tension according to Schiötz is shown in table 2. After that the patient was not seen any more.

CASE 16.—J. W., aged 14, was seen two and one-half hours after contusion of the left eye by a small piece of wood.

The right eye had normal visual acuity, with hypermetropia of 0.5 D.

The left eye had visual acuity of 1/3; vision was normal with a -0.75 sphere. Visual acuity in this eye later returned to normal without glasses.

TABLE 2.—*Tension in Millimeters for Case 15*

Date	Right Eye	Left Eye	Difference
7/31/35.....	23.0	20.0	+ 3.0
8/ 1/35.....	19.0	25.5	- 6.5
8/ 2/35.....	15.0	25.5	-10.5
8/ 5/35.....	19.0	22.0	- 3.0
8/ 7/35.....	15.5	19.0	- 3.5

TABLE 3.—*Tension in Millimeters for Case 16*

Date	Right Eye	Left Eye	Difference
7/ 7/35.....	17.0	16.0	- 1.0
7/ 8/35.....	19.0	7.5	-11.5
7/10/35.....	17.0	8.5	- 8.5
7/12/35.....	15.5	8.5	- 7.0
7/15/35.....	18.0	8.5	- 9.5
7/17/35.....	18.0	12.0	- 6.0
7/19/35.....	20.5	13.0	- 7.5
7/22/35.....	22.0	14.0	- 8.0
7/26/35.....	19.0	15.0	- 4.0
7/31/35.....	19.0	18.0	- 1.0

TABLE 4.—*Tension in Millimeters for Case 17*

Date	Right Eye	Left Eye	Difference
11/16/35.....	17	25.5	+8.5
11/17/35.....	17	19.0	+2.0
11/18/35.....	19	22.0	+3.0

There were slight abrasions of the corneal epithelium. The anterior chamber was deep, and the aqueous was slightly turbid (the next day a small hyphemia was seen). The pupil was slightly contracted and angular and showed no reaction to light. The fundus showed a white spot of Berlin's edema in the upper portion.

The tension according to Schiötz is shown in table 3.

CASE 17.—P. R., aged 38, was seen three or four hours after contusion of the left eye (he struck his head against a bar of iron).

Tests of visual acuity and refraction were not carried out. However, there were no clinical signs of predisposition to glaucoma.

The left eye showed a small tear of the bulbar conjunctiva and erosion of the cornea. Otherwise, there were no changes.

The tension according to Schiötz is shown in table 4.

CASE 18.—H. K. L., aged 26, was seen half an hour after contusion of the left eye by a fragment of brass.

Visual acuity was normal in each eye, and the eyes were emmetropic.

The left eye showed a small wound of the conjunctiva and erosion of the cornea; otherwise, it was normal.

The tension according to Schiötz is shown in table 5.

CASE 19.—J. V., aged 41, was seen one-half hour after contusion of the right eye by a hammer.

Visual acuity of each eye was 1 partly; with a +0.5 sphere it was normal.

The right eye showed a small hemorrhage under the bulbar conjunctiva. The pupil was slightly larger and reacted more slowly to light than that of the left eye. Otherwise, no changes were seen.

The tension according to Schiötz is shown in table 6.

TABLE 5.—*Tension in Millimeters for Case 18*

Date	Right Eye	Left Eye	Difference
12/11/35.....	17.0	15	—2.0
12/12/35.....	17.0	12	—5.0
12/13/35.....	15.5	13	—2.5
12/16/35.....	13.0	13	Equal

TABLE 6.—*Tension in Millimeters for Case 19*

Date	Right Eye	Left Eye	Difference
12/24/35.....	15.5	20.5	—5
12/27/35.....	15.5	20.5	—5

TABLE 7.—*Tension in Millimeters for Case 20*

Date	Right Eye	Left Eye	Difference
1/ 9/36.....	27.5	27.5	Equal
1/10/36.....	27.5	19.0	—8.5
1/11/36.....	27.5	24.0	—3.5
1/13/36.....	27.5	20.5	—7.0

CASE 20.—W. G. O., aged 7, was seen one hour after contusion of the left eye by a stick.

On the second day after the accident visual acuity was normal in each eye, with hypermetropia of 1 D.

The left eye showed (on the day of the accident) erosion of the cornea; otherwise, there were no changes.

The tension according to Schiötz is shown in table 7.

CASE 21.—P. V., aged 16, was seen one-half hour after contusion of the left eye by a fragment of iron.

Visual acuity was normal; there was hypermetropia of 0.5 D.

Examination of the left eye revealed erosion of the cornea. The pupil was slightly oval and was slightly more contracted than the pupil of the other eye. Otherwise, no changes were seen.

The tension according to Schiötz is shown in table 8.

CASE 22.—H., aged 37, was seen one-half hour after contusion of the left eye by an accidental kick.

The left eye showed a small wound of the eyelids and erosion of the cornea. The pupil was obliquely oval and showed only a slight reaction to light. Temporally and above, Berlin's edema of the retina was seen. There were no tears and no hemorrhage.

On the following day visual acuity was normal in the right eye (the eye was emmetropic); visual acuity in the left eye was $1/2$, and with a $+0.5$ D. cylinder it was normal.

The tension according to Schiötz is shown in table 9.

TABLE 8.—*Tension in Millimeters for Case 21*

Date	Right Eye	Left Eye	Difference
1/10/36.....	20.5	15.5	—5
1/11/36.....	20.5	15.5	—5
1/17/36.....	20.5	20.5	Equal

TABLE 9.—*Tension in Millimeters for Case 22*

Date	Right Eye	Left Eye	Difference
1/22/36.....	15.5	18.0	+2.5
1/23/36.....	20.5	15.5	—5.0
1/24/36.....	20.5	17.0	—3.5

COMMENT

Summarizing the clinical cases of traumatic glaucoma, one finds the following:

In the third case there were subluxation of the lens, a torn and atrophied iris, a hemorrhage into the vitreous, a hole at the macula and a rupture of the choroid and the retina close to the ora serrata. Visual acuity was movements of the hands only.

In the fourth case there were hemorrhage into the anterior chamber and the vitreous, atrophy of the iris with peripheral anterior synechiae, rupture of the choroid and retina near the ora serrata and a slight injury of the macula lutea. Visual acuity was $1/3$.

In the fifth case the aqueous was sticky, and there were a hemorrhage into the vitreous and peripheral rupture of the retina and perhaps also of the choroid. Visual acuity was normal.

In these cases it was necessary to operate. The result was satisfactory, as the impaired visual acuity in the third and fourth cases is explained by the injury to the macula lutea. It will be noted that in all these cases a hemorrhage into the vitreous had occurred, in one case combined with dislocation of the lens. In the following cases these serious injuries were absent, and conservative treatment gave satisfactory results.

In the sixth case there was an attack of acute glaucoma.

In the seventh case there was an attack of acute glaucoma without visible injuries to the inner part of the eye. The curve for tension of this eye shows great similarity to the curves in the cases of simple contusion, in most of which the eye showed slight injuries.

Summarizing the cases of simple contusion, one finds that in fifteen cases slight intra-ocular lesions and a varying degree of instability of tension were present. In case 12 hypotony was combined with subluxation of the lens. In the literature dislocation of the lens is regarded as one of the most important causes of glaucoma associated with dislocation of the lens. This theory is erroneous, as will be discussed later (see "Conclusions"). Instability of tension without any accompanying visible intra-ocular injury (in a few instances in this series the pupil

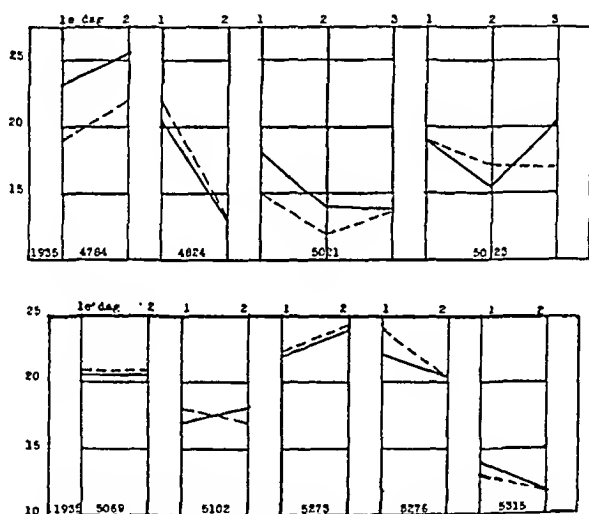


Fig. 30.—Diagrams showing tension in each eye in nine control cases. The broken line indicates the tension in the injured eye, and the continuous line, the tension in the sound eye.

was slightly larger than that in the sound eye) was observed in seven cases (10, 14, 17, 18, 19, 20 and 21). In case 14 (the patient was examined on the second day) and in case 17 (the error of refraction was unknown) a few details are missing, but in the remaining five cases the clinical examination was complete.

On comparing all the cases of traumatic glaucoma (anatomic and clinical) one finds that in two cases (1 and 2) severe intra-ocular lesions were present, and the eye had to be removed (see "Anatomic Part"); that in two other cases (3 and 4) severe lesions were present but the eye was saved, although visual acuity was reduced; that in yet two other cases (5 and 6) the lesions were much less severe, and vision returned to normal. In the last case (7) there was no intra-ocular lesion. It is remarkable that the curve for tension in this case resembles the curves found in cases of simple contusion.

In the cases of simple contusion slight injuries were found in one group and no injuries in another. Yet in all the cases there was instability of tension.

From this comparison one may conclude that from the most severe cases of traumatic glaucoma to the simplest cases of contusion every intermediate stage is found in two respects: (*a*) as regards the severity of the intra-ocular injury and (*b*) as regards the severity of the instability of tension.

One may also conclude that instability of tension may occur in young patients with normal eyes after contusion causing no visible injury to the inner part of the eye.

As it seemed possible that the dressing applied to the eye might influence the intra-ocular tension, nine control cases were observed. No marked influence could be ascertained.

III. ANALYSIS OF CASES REPORTED IN THE LITERATURE

In this chapter all the cases of traumatic glaucoma reported in the available literature are considered, together with the cases described in the two previous sections. Other cases have been added which were not described as instances of traumatic glaucoma but which may be regarded as such.

These cases were grouped according to the accuracy of the description as follows:

I. Reliable cases (refraction between hypermetropia of 3.5 D. and myopia of 10 D.).

II. Less reliable cases (refraction not known).

The reliable cases and the unreliable cases were grouped according to the result of treatment as follows:

(*a*) Cases in which the result of treatment was favorable: tension normal; visual acuity $1/2$ or more.

(*b*) Cases in which the result of treatment was less favorable: tension normal; visual acuity from $1/60$ to $1/3$.

(*c*) Cases in which the result of treatment was unfavorable: tension raised or visual acuity less than $1/60$ or eye removed.

The old figures indicating errors of refraction were recalculated in diopters, and the figures indicating visual acuity were reduced to simple fractions.

The third and fourth groups are unsuitable for study and may be dismissed without discussion.

III. Cases without (sufficient) obtainable data. These were reported by the following authors: Aubineau (1910), Chalupecky (1905).

TABLE 10.—Data for Reliable Cases in Which the Result of Treatment Was Favorable (Group I a)

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes				Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemor- rhage	Iris	Lens	Retina	Choroid	Glau- coma	First Treat- ment			
Bralley, 1883.....	14	Oork	Hyperme- tropia -2	Cataract	2 days	8 days	Physostigmine	T.N.; V. = 0.5	
Ferber, 1887.....	?	Piece of iron	Hyperme- tropic astig- matism -2	Hemor- rhage and exudation	3 wks.?	Physostigmine; Iridectomy	T.N.; V. = 0.5 (partly)	
Knapp, 1883.....	29	Stick	Emmetropia?	Anterior disloca- tion	12 days	A few days	Physostigmine	T.N.; V. = 1 with glasses	At first hypotony; later, hypertony, with normal ante- rior chamber and turbid vitreous
Lavat, 1924.....	41	Flst	Hyperme- tropic astig- matism -1.5	Corpus vitreum	?	18 days	Pilocarpine; Iridectomy; trep- anation; para- centesis	T.N.; V. = 1 with glasses	Optic nerve cupped
Magitot, 1917, first case	34	Explosion	Emmetropia?	?	1 day	T.N.; V.: Right eye, %; left eye, 1	
Magitot, 1917, second case	?	Fall	Myopic astig- matism +0.5	Slight venous dilatation	?	? hrs.	T.N.; V. = % with glasses	
Magitot, 1917, third case	32	Indreet	Emmetropia?	?	4 days	T.N.; V. = 1	
Magitot, 1917, fourth case	?	Lump of clay	Myopia +1	Anterior chamber	?	5 days	T.N.; V. = 1 with glasses	
Magitot, 1918, first case	24	Fall	Emmetropia?	?	2 days	T.N.; V. = 1	Fracture of orbit
Marx and Quartero, 1930	13	Piece of coal	Myopic astig- matism +0.75	Anterior chamber	Trembling	Tears; ablatio	21 days	Atropine; pilocarpine	T.N.; V. = 1 with glasses	At first, hypotony; atropine discon- tinued on twenty- first day
Motolosc, 1912.....	?	?	Hyperme- tropia +2.5	Anterior chamber shallow	?	?	Physostigmine	T.N.; V. = 0.6	

Myers, 1901.....	?	Stone	Astigmatism	Anterior chamber; corpus vitreum	Subluxation	Comotio	Subretinal hemorrhage	5 days	7½ wks.	Atropine; physostigmine; pilocarpine	T.N.; V. = 1 with glasses	Atropine replaced by miotics on fifth day because of slightly raised tension; in third week attack of acute glaucoma with deep anterior chamber
Peters, 1904, first case	29	Piece of wood	Hypermetropia -1	Cataract	?	18 days	4 wks.	Physostigmine	T.N.; V. = 5/6 with glasses	
Peters, 1904, second case	57	Whip	Emmetropia?	Anterior chamber	7 days	Atropine; physostigmine; paraentesis	T.N.; V. = 5/7	Atropine administered during 8 days
Pye-Smith, 1882.....	70	Fall	Hypermetropia -1.5	?	?	?	?	?	3 days?	5 days	21 mos.	Physostigmine	T.N.; V. = 1 (partly)	
Sala, 1904, first case....	53	Cork	Hypermetropia -1.25	Anterior chamber; corpus vitreum	?	?	3 mos.	Paraentesis	T.N.; V. = 5/7	
Sala, 1904, second case	38	Stone?	Hypermetropia -3.5	Anterior chamber	5 days	6 wks.	Scopolamine; physostigmine; paraentesis	T.N.; V. = 1	Because of hypertony scopolamine given until fifth (?) day
Scheffels, 1891.....	35	Stick	Myopia 1%	Anterior chamber; corpus vitreum	Dialysis	Cataract	Detachment	2 days	12 wks.	Atropine; physostigmine	T. + ?; V. = 2%	After 12 years tension normal; total cataract; visual acuity 1/60
Tillemma, fifth case.....	35	Belt	Hypermetropia astigmatism -0.5	Corpus vitreum	Small tear	3 days	3 days	7 days	Physostigmine; iridectomy	T.N.; V. = 1 with glasses	Anterior chamber remains deep after having been opened
Tillemma, seventh case...	54	Piece of wood	Emmetropia	Hours	2 days	Pilocarpine	T.N.; V. = 1	
Villard, 1905, first case	66	Piece of wood	Hypermetropia -1	12 days	4 days	Physostigmine	T.N.; V. in right eye and left eye, 1/3	
Villard, 1906, third case	63	Wooden stopper	Myopia +5	Anterior chamber	Tear	Trembling	4 hrs.	4 days	Physostigmine; epinephrine; pilocarpine	T.N.; V. = 0.3 with glasses	Other eye: visual acuity with glasses, 0.4

* T. Indicates tension; T.N., normal tension, and V. vision.

TABLE 11.—Data for Reliable Cases in Which the Result of Treatment Was Less Favorable (Group 1 b)

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes				Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result* T.N.	Comment
				Hemor- rhage	Iris	Lens	Retina	Choroid	Glau- coma	First Treat- ment			
Agnello, 1931, second case	75	Branch of tree	Emmetropia	?	?	?	?	?	3 days	3 mos.	Iridectomy	T.N.	Traumatic myopia of 3.5 D.
Bourgeois, 1904, first case	32	Cork	Emmetropia	Anterior chamber	Anterior subluxation	1 day	1 day	Pilocarpine	T.N.; V. = 0.25 with glasses	
Brand, 1903.....	30	Fragment of steel	Hypermetropia -2.5	5 days	Atropine; scopolamine; physostigmine; iridectomy	T.N.; V. = 1/3	Physostigmine administered when optic nerve was already cupped
Darier, 1899.....	46	Piece of wood	Emmetropia?	?	?	Anterior chamber very shallow	?	?	4 wks.	A few days?	Physostigmine; iridectomy	T.N.; V. = 1/3 with glasses	Traumatic myopia of 3.5 D.; other eye emmetropic, with visual acuity of 1
Fromaget, 1913, second case	25	Piece of wood	Hypermetropic astigmatism -1.5	Anterior chamber	Trembling	2 hrs.	Pilocarpine	T.N.; V. = 1/3	
Herrmann, 1906.....	47	Iron nut	Hypermetropia -2.25	Corpus vitreum?	Cataract; anterior chamber shallow	?	?	4 days	Physostigmine; iridectomy	T.N.; V. = 4/60	
Piek, 1904.....	40	Jet of water	Emmetropia	Corpus vitreum	Cataract	Equatorial choroiditis	Weeks or months	1 yr.	Pilocarpine; iridectomy	T.N.; V. = 1/2	Shallow, gray excavation of optic nerve
Stoewer, 1904.....	23	Stick	Emmetropia?	Anterior chamber; corpus vitreum	Cataract	Small scar	4 days	2 days	Proeaine borate; pilocarpine; physostigmine; sclerotomy twice; iridectomy	T.N.; V. = 4/60	Optic nerve slightly atrophic
Stoewer, 1907.....	65	Explosion	Hypermetropia -3.5	?	?	?	?	?	?	?	Iridectomy	T.N.; V. = 4/35	Central old macula corneae

* T. Indicates tension; T.N., normal tension, and V. vision.

TABLE 12.—*Reliable Cases in Which the Result of Treatment Was Unfavorable (Group 1c)*

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemor- rhage	Iris	Lens	Retina	Choroid	Glau- coma	First Treat- ment				
				Anterior chamber; corpus vitreum	Atrophy	Macular lesion	Peripheral tear	4 days	1 day	1 day	Atropine; pilocarpine; paracentesis; iridectomy	T.N.; V. = $\frac{1}{2}$ with glasses	
Tillemann, fourth case...	34	Brass wire	Hypermetropia -0.5	?	?	?	?	?	10 days	8 days	?	Miotics; iridectomy	T.+; enucleation elsewhere	Fracture of orbit
Aguello, 1931, first case	54	Fist	Hypermetropia astigmatism	Anterior chamber	Tear	1 day?	2 days?	6 wks.	Pilocarpine; physostigmine; paracentesis	T.N.; V. = perception of light	
Magtrot, 1917, fifth case	41	Branch of tree	Emmetropia	?	Detachment	Subluxation?	?	?	9 wks.	3 days	Physostigmine	T.+2; V. = 0; enucleation	
Priestley Smith, 1881...	57	Cork	Hypermetropia -2.5	Corpus vitreum	Temporal subluxation	Not visible	Not visible	3 days	6 days	14 wks.	Physostigmine; iridectomy	Enucleation	Immediately after iridectomy, enucleation because of prolapse of vitreous
Priestley Smith, 1883...	59	Fist	Hypermetropia -1.5											
Tillemann, first case.....	26	Fall	Emmetropia	Anterior chamber; corpus vitreum	Tear	Cataract	Tear	Tear	25 days	11 hours	3 wks.	Pilocarpine	T.+; V. = perception of light; enucleation	
Tillemann, second case...	30	Piece of wood	Emmetropia	Corpus vitreum	Tear	Capsule torn; luxation	Tear	Tear	7 days?	Hours	7 wks.	Pilocarpine	T.+; V. = perception of light; enucleation	Patient refused regular treatment
Tillemann, third case.....	41	Piece of brass	Hypermetropia -0.5	Anterior chamber; corpus vitreum	Tear	Subluxation	Tear; hole at macula	Tear	6 days	Hours	18 days	Pilocarpine; extraction of lens	T.N.; V. = $\frac{2}{300}$	

* T. indicates tension; T.N., normal tension, and V. vision.

TABLE 13.—Data for Less Reliable Cases in Which the Result of Treatment Was Favorable (Group II a)

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Returned After	Treatment	Result* Complete cure	Comment
				Hemor- rhage	Iris	Lens	Retina	Choroid	Glau- coma	First Treat- ment				
Armaignae, 1907.....	?	Piece of wood	?	?	?	?	?	?	?	?	?	Operation	Complete cure	
Augustin, 1904.....	?	?	?	?	?	?	?	?	?	?	?	Physostigmine	Favorable	
Beauvois, 1908.....	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	T.N.	
Boucheron, 1880.....	Young	Snowball	?	Corpus vitreum	?	8 days	3 days	Paracentesis six times	T.N.; V. = same in both eyes	
Cantonnnet, 1912.....	49	Piece of concrete	?	?	1 day	5 days	Cocaine; pilocarpine; atropine physostigmine	T.N.; V. = 1	Atropine with ear for tension of -2
Cohn, 1904.....	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	Favorable	
Cramer, 1931, second case	42	Piece of iron	?	Anterior chamber	?	?	?	?	2 days	?	?	Paracentesis	T.N.	
Fromaget and Fromaget, 1913, first case	35	Iron chain	?	Anterior chamber; corpus vitreum	14 days?	?	Pilocarpine; physostigmine iridectomy	T.N.	
Fromaget, 1924.....	?	?	?	?	?	?	?	?	?	?	?	Miotics; epinephrine and procaine hydrochloride retrobulbarly	Favorable	
Guilbert, 1922, first case	64	Cork	?	Anterior chamber	?	?	?	?	?	?	14 days	Pilocarpine; sodium iodide subconjunctivally	T.N.; V. = 2/3	Optic nerve paler than that in other eye
Guilbert, 1922, second case	58	?	?	?	?	?	?	?	?	18 days	14 days	Pilocarpine; sodium iodide subconjunctivally	T.N.; V. = 1	
Guilbert, 1922, third case	59	?	?	?	?	?	?	?	?	?	2 days	Pilocarpine; sodium iodide subconjunctivally; iridectomy	Favorable	
Jacobson, 1884, first case	67	Wooden stopper	?	Corpus vitreum?	Anterior chamber shallow; cataract	?	?	?	8 days	2 days	Atropine; paracentesis	T.N.; V. = 2/3	Paracentesis = unfinished iridectomy

TABLE 14.—Data for Less Reliable Cases in Which the Result of Treatment Was Less Favorable (Group II b)

Author	Age, Years	Trauma from	Refraction	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilibrium Returned After	Treatment	Result*	Comment
				Hemor- rhage	Iris	Lens	Anterior dislocation	Retina	Choroid	Glau- coma	First Treatment			
Bourgeois, 1904, second case	69	Fall	?	?	5 wks.	Pilocarpine; iridectomy	T.N.; V. = $\frac{1}{6}$ with glasses	Traumatic myopia of 3 D
Cordes and Horner, 1932, first case	18	Tomato	?	Anterior chamber; corpus vitreum	5 days	?	Ethymorphine hydrochloride; atropine; physostigmine; paracentesis	T.N.; V. = 0.4 with glasses	Patient discharged on fourth day; hemorrhage into vitreous and glaucoma on fifth day after playing football
Hirschberg, 1883, first case	15	Bits of wood and iron	?	Corpus vitreum	Tear	Subluxation	Macular lesion	Tear	7 days	3 wks.	Atropine; physostigmine; iridectomy	T.N.; V. = $\frac{15}{200}$	Atropine not administered by Hirschberg
Jacobson, 1884, second case	64	Piece of wood	?	Luxation	Halo glaucomatosus	4 days	4 wks.	Paracentesis	T.N.; V. = 0.1	Paracentesis-unfimbriated iridectomy
Mellinger, 1899	26	Iron chain	?	?	?	?	?	?	?	Sev- eral days	?	Iridectomy	T.N.; V. = reduced	
Müller, 1895	44	Branch of tree	?	Corpus vitreum	Luxation in corpus vitreum	?	?	?	14 days	Physostigmine; paracentesis	T.N.?, V. = $\frac{2}{60}$ with glasses	Paracentesis-unfimbriated extrac-tion of lens
Padovani, 1929	54	Stone chip	?	?	?	?	?	?	?	30 days	?	Pilocarpine	T. lower (normal?)	
Sebindhelm, 1917 first case	41	Cow's horn	?	Anterior chamber	Tear	Cataract	?	?	?	-?	3 days?	Physostigmine	T.N.; V. = $\frac{5}{12}$?	
Siegfried, 1896	18	Iron nut	?	Anterior chamber; corpus vitreum	Hemor- rhage	2 hrs.	?	T.N.?, V. = ability to read Jaeger's test type no. 15	Gelatinous exudate in anterior chamber
Villard, 1905, third case	6	Toy pistol	?	Anterior chamber	-?	-?	-?	Detach- ment	-?	4 days	7 days	Physostigmine; pilocarpine; ethymorphine hydrochloride	T.N.; V. = 0.1	Detachment of retina cured; atrophy of optic nerve
Spencer Watson, 1880....	65	Stone	?	Anterior chamber	Trembling	?	?	?	?	10 days	Physostigmine; sclerotomy	T.N.; V. = ability to read Jaeger's test type no. 20	

* T. Indicates tension; T.N., normal tension, and V. vision.

Fischer (1908), Genet (1922 and 1925), Grand-Clément (1911), Hansell (1902), Helberg (1906), Klauber (1922), Krienes (1895), de Schweinitz (1909) and Wagenmann (1915).

IV. Cases in which predisposition to glaucoma seemed likely (because of hypermetropia); cases in which there was myopia of more than 10 diopters or other abnormalities. These were reported by the following authors: Agnello (1931), Agnew and Webster (1886), Bourgeois (1904), Burchhardt (1896), Deutschmann (1916), Glauning (1902), von Hippel (1900), Landesberg (1869), Mazza (1907), Oliver (1896), Reid (1929), Salzer (1915), Scheer (1903), Schindhelm (1917), Schmidt-Rimpler (1883), Sédan (1925) and Thilliez (1906).

COMMENT ON CLINICAL DATA

The total number of one hundred and six cases, to which may be added a certain number without sufficient data, is small for the number of years over which they have been collected. One must not be too dogmatic, therefore, in drawing conclusions. One would be inclined, for instance, to think that in the majority of cases the course is favorable. This would be an erroneous view, as few cases in which there was dislocation of the lens have been included in the tables, while experience has shown that in such cases the course is usually unfavorable. On the other hand, the tables probably also give a wrong impression of the cases in which the course was favorable because many cases in which the intra-ocular tension was only slightly raised must have escaped notice. My cases of "simple contusion" have convinced me that the intra-ocular tension must be checked tonometrically to find a difference in tension between the two eyes.

Traumatic glaucoma apparently occurs at every age. The extremes are 6 years (Villard, 1905, third case) and 75 years (Agnello, 1931, second case). This is easily understood, as beyond these age limits one is not so much exposed to trauma.

The objects that cause glaucoma vary greatly in size and in weight. A common feature, however, is the fact that they strike the eye with great velocity (e. g., the fist, a cricket-ball).

In seventy-four cases the time of onset of the attack was mentioned. In thirty-two of these the attack came on immediately after contusion; in twenty-one other cases, after from three to five days, and in six cases, a little later, but within the first week. In fifty-nine cases, therefore, glaucoma occurred in the first week. In only six cases did it occur in the second week. In two cases it occurred in the third week, and in the remaining cases still later, up to seven weeks. One may say, therefore, that in the majority of cases glaucoma occurs in the first week after contusion.

TABLE 15.—Data for Less Reliable Cases in Which the Result of Treatment Was Unfavorable (Group II c)

Author	Age, Trauma Years from	Refraction	Hemor- rhage	Intra-Ocular Changes					Period Between Trauma and		With Treatment Equilib- rium Returned After	Treatment	Result*	Comment
				Anterior chamber	Iris	Lens	Retina	Choroid	Glau- coma	First Treat- ment				
Augstein, 1904	?	?	?	?	?	?	?	?	?	?	?	Iridectomy	Poor	Partial rupture of sclera
Buehanan, 1907	59 Broomstick	?	?	?	?	Disloca- tion	?	?	?	?	6 mos.	Enucleation	
Cabannes, 1908, first case	38 Book	?	Subluxa- tion	?	?	3 days	?	Months	Sclerotomy twice; phloeoc- raphy; iridectomy impossible	T. +2; V. = ?	
Cabannes, 1908, second case	60 Cork	?	Anterior chamber	Tear	?	?	?	?	?	Iridectomy	T.N.; V. = al- most blind	Six months later, cataracta trau- matica
Cordes and Horner, 1932, third case	35 Whip	?	Anterior chamber	?	?	?	?	4 days	?	More than 13 days	Atropine; para- centesis twice	V. = 0	
Cramer, 1931, third case	16 ?	?	Anterior chamber; corpus vitreum?	?	?	?	?	?	6 days	?	?	Paracentesis	T.N.; V. = ?	Two months later, detachment of retina
Cramer, 1931, fourth case	25 Iron chain	?	?	?	?	?	?	?	5 days	?	6 mos.	Paracentesis	T. = 30; V. = 0	Patient did not follow instructions
Cramer, 1931, fifth case	61 Piece of wood	?	?	?	?	?	?	?	-?	?	Months	Iridectomy; miotics; trepa- nation; para- centesis	T.N.; V. = 0	
Cramer, 1931, sixth case	59 Piece of wood	?	?	?	?	Subluxa- tion	?	?	?	?	?	1% physostig- mine in live oil; iridectomy	T. = 50; V. = ½	
Erdmann, 1900.....	66 Piece of wood	?	?	?	?	Anterior disloca- tion	?	?	-?	4 wks.	3 days	V. = perception of light; enuclea- tion	
Fromaget, 1907	35 Iron chain	?	?	?	?	?	?	?	14 days	Hours	More than 5 wks.	Atropine; phloocarpine; physostigmine; iridectomy	T. +; V. = 1/10?	Atropine discon- tinued on sixth day; rupture of sclera

Garnier, 1891	14 Stick	?	Corpus vitreum	Trembling	?	?	3 days	12 days	Atropine; physostigmine	Enucleation	Atropine discontinued on third day; partial rupture of sclera?
Goldzieher, 1916	?	?	?	Cataract	Fine peripheral pigmentation	2 mos.?	?	?	?	T. = from 40 to 50	Optic nerve deeply cupped
Hirschberg, 1883, second case	38 Piece of iron	Myopia?	?	Hemorrhagic infiltration	3 mos.	A few days	?	Atropine; physostigmine; iridectomy refused	T. +1	Immediately after contusion T. -1; atropine administered; patient dismissed on second day
Hoor, 1888	?	?	Corpus vitreum?	?	?	Detachment	?	?	?	More than 3 mos.	Pilocarpine; physostigmine; iridectomy	T. +	
Lamb, 1927	9 Rope	?	Anterior chamber	Tear	Cataract	?	?	?	?	8 wks.	?	Enucleation	
Martin, 1907, 3 cases....	?	?	?	?	?	?	?	?	AV. already = 0	Glaucoma absolutum	
Mezzatesta, 1929	76 Fist	?	?	?	?	?	?	3 days	?	?	T. +; V. = 0	
Morax, 1921, seventeenth case	27 Fist	?	Cataract	A few days?	2 yrs.	20 mos.	Extraction of lens; trepanation; sclerectomy; iridectomy	T. = 10; V. = 0	
Morax, 1921, eighteenth case	65 Jet of water	?	Anterior chamber	?	Anterior dislocation	Hemorrhage	Hemorrhage	1 day	1 day	4 wks.	Pilocarpine; physostigmine; anterior sclerotomy	V. = 0; enucleation	
Scalzititi, 1927	19 Tennis ball	?	?	?	?	Detachment	Pigmentation	30 days	?	?	Miotics; repeated paracentesis	T.N.; A. = 0	
de Schweinitz, 1893	46 ?	?	?	?	?	Papillitis	?	From 7 to 22 days	7 days?	More than 3 mos.	Physostigmine	T. = + 1; V. = 0	Patient did not follow instructions
Thilbert, 1920	33 Fragment of wood	?	?	?	?	?	?	12 or 13 days	13 days?	More than 2 mos.	Pilocarpine	T. = 65	
Villard, 1905, second case	52 Piece of concrete	?	-?	-?	-?	-?	-?	-?	10 days	?	Physostigmine	T. +; V. = 0	Patient refused operation

* T. indicates tension; T.N., normal tension, and V. vision.

In checking the percentage of intra-ocular lesions, tears of the iris and intra-ocular hemorrhage have been grouped together, as a tear of the iris is always accompanied by hemorrhage. Pathologic changes of the choroid and the retina have also been grouped together, as they usually accompany each other. Many cases were found to lack so many details that it seemed inadvisable to use them. In table 16 only the data for the reliable cases have been used, and among these only those cases have been considered in which the required item was reported. Thus this table gives an idea of the relation between the course of the disease and the intra-ocular lesions.

It is found, therefore, that the number of intra-ocular hemorrhages, dislocations of the lens and pathologic changes in choroid and retina run parallel with the course and severity of the disease. Unfortunately,

TABLE 16.—*Relation Between Course and Intra-Ocular Lesions*

	Incidence	Percentage
Hemorrhage		
23 cases with favorable course.....	12	50
8 cases with less favorable course.....	6	75
6 cases with unfavorable course.....	6	100
Dislocation of the lens		
23 cases with favorable course.....	5	22
8 cases with less favorable course.....	3	37
6 cases with unfavorable course.....	4	66
Changes in the choroid and retina		
22 cases with favorable course.....	7	33
6 cases with less favorable course.....	3	50
4 cases with unfavorable course.....	3	75

the number of cases is too small to give these figures absolute value, but it supports my opinion, which will be discussed later, that dislocation of the lens is accompanied by other severe intra-ocular damage and constitutes a symptom of the disease but not its cause. If the course is unfavorable in cases in which there is dislocation of the lens, one may conclude from the dislocation of the lens that other serious lesions are present. The dislocation of the lens possesses, therefore, only prognostic significance.

It was found impossible to draw conclusions from the length of the period that elapsed between the onset of the disease and the first treatment, as the figures are too much influenced by the data for one or two particular cases.

IV. CONCLUSIONS

ORIGIN—PROGNOSIS—TREATMENT

ORIGIN

Although reports of reliable cases of traumatic glaucoma were published as early as 1881 (Priestley Smith), few more than a hundred instances can be collected from the literature. In many other cases

the condition has been labeled "glaucoma through dislocation of the lens," a term which is attractive but misleading, as will be discussed later. Anatomically, little was known about traumatic glaucoma as the eye had been examined in only two cases (Garnier, 1891, and Morax, 1922). Recently I added two more cases (first section). Experiments on contusion in animals have been carried out several times, but although they yielded interesting information they failed to reproduce traumatic glaucoma.

Consequently the present knowledge of this subject is limited. Theories as to the origin of this traumatic rise of tension cover the whole field of glaucoma, but their value is proportional to the number of cases (usually only a few) observed by the authors. Nevertheless, this value is enhanced by the small total number of observations recorded. For this reason they may be briefly reviewed here. The following opinions have been expressed from time to time:

1. Traumatic glaucoma, if it occurs at all, must be uncommonly rare (A. von Graefe, 1869).

2. Traumatic glaucoma does not exist; all cases representing those of predisposed patients (Lagrange, 1922).

According to other writers, glaucoma is caused by:

3. Dislocation of the lens (C. and H. Fromaget and Thiel). This cannot be the cause, as glaucoma may occur after the lens has been expelled (Morax).

4. Accumulation of fluid in the choroid through blocking of the perivascular lymphatics with loose cells (Garnier).

5. Tear of the ciliary muscle, interfering with filtration at the angle of the anterior chamber (Garnier and Stoewer).

6. Pushing of the iris into a rupture of the sclera, with closing of the angle of the anterior chamber (Fuchs).

7. Rise of the albumin content of the aqueous humor through vascular paralysis (Peters and Sala). This is impossible, as the rise of pressure precedes the rise of the albumin content (Leplat and Magitot).

8. Thrombosis of an artery in the ciliary body (Garnier).

9. Derangement of the vascular innervation (Garnier), causing edema of the ciliary body with displacement of the angle of the anterior chamber (Stoewer).

10. Irritation of a sympathetic nerve fiber, causing hypersecretion and obstruction of filtration through vasoconstriction (C. and H. Fromaget), or causing variation of the volume contents of the eye (Marx and Quartero).

11. Vasomotor reactions through influence of the sympathetic nervous system (Magitot, Leplat and Schmidt and de Decker).

12. "Passive hypertony" through obstruction of the orbital circulation caused by an orbital hematoma (Magitot). This theory is not convincing (see later comment).

13. Hemorrhage (Morax).

As there is no consensus on this point, the subject is well worth further study.

Experiments (Leplat, Schmidt and de Decker and others) have demonstrated that in animals contusion of the eyeball causes a short period of hypertony followed by a longer period of hypotony but that glaucoma does not supervene. The tension readjusted itself in a few days even after a powerful blow.

Cases reported by Leplat, Magitot and myself show that in man also contusion is followed by variations of the intra-ocular tension. It is difficult to say whether hypertony usually occurs immediately after contusion, to be followed later by hypotony, or whether hypotony occurs at once without hypertony. Obviously our data lack accuracy, as one dares not measure the tension more than once each day, while in animals one can take a reading every few minutes. Suffice it to state that in a number of cases after a simple contusion I found a rise of tension followed by a fall of tension but that in another series of cases I found only hypotony. In every case, however, the intra-ocular tension in the affected eye differed from that in the sound eye for a number of days, sometimes for weeks. In the end the tension returned to normal.

In a number of other cases glaucoma supervened. Why did the tension return to normal in some cases? Why not in every case? What was the difference between these two groups of cases? One must endeavor to deduce this difference from the anatomic and clinical evidence.

The difference between these two groups of cases, those of traumatic glaucoma and those of simple contusion, is obvious. Serious intra-ocular lesions associated with glaucoma were present in the one group and insignificant lesions with only hypertony or hypotony in the other. These lesions, therefore, must give the key to the problem.

Before discussing them, however, other possibilities must be viewed. It has been stated by Lagrange that traumatic glaucoma does not exist, all patients with glaucoma following trauma representing patients who are predisposed to an attack of glaucoma. It is, of course, impossible to exclude this possibility in all cases. Yet it is difficult to accept Lagrange's opinion if one recollects that in almost every case contusion is followed by variations of tension. It is also difficult to believe that traumatic glaucoma in childhood is due to predisposition, as other forms of glaucoma are excessively rare in this period of life. However, it is

well to keep this possibility in mind when it is necessary to operate on older patients and to perform iridectomy in preference to paracentesis of the anterior chamber.

Magitot (1917-1918) expressed the opinion that the globe, although normal, may suffer from passive hypertony through obstruction of the extrabulbar drainage channels by an orbital hematoma. However, the globe had been examined from two to five days after the accident. In every instance the globe may have been injured, as a slight intra-ocular hemorrhage may easily be absorbed in that period. The fact that in some cases hypertension and palpebral hematoma declined together may have been accidental. Furthermore, the presence of a palpebral hematoma combined with normal movements of the globe (1917, first and fourth cases) hardly proves that an orbital hematoma or edema is present. Moreover, in the fourth case, which has just been mentioned, hemorrhage into the iris was seen, which proves that the globe had been injured. In another case published in 1918 (first case), the patient was examined two days after contusion. Finally, in one case (1917, seventh case) in which there was a fairly large hematoma of the eyelids, hypotony was noted instead of hypertony. Therefore, the theory of passive hypertony lacks sufficient evidence, and all these cases must probably be classified with those of other authors in which there was active hypertony.

If one cannot accept predisposition or retrobulbar obstruction as a factor, glaucoma must be caused by the traumatic lesions which one observes in nearly every case. An intra-ocular hemorrhage may be a bar to normal filtration, as in cases of hyphemia after operations for cataract. A late hemorrhage also may exert a bad influence on the course of events, as is illustrated by my fourth case and by Cordes and Horner's cases. This cannot be a frequent cause, as many investigators noticed that glaucoma occurred after the blood had been resorbed (e. g., my sixth case). In my first case also the tension rose a long time after the second hemorrhage had been resorbed. Moreover, glaucoma may occur without hemorrhage.

It is unlikely that iridodialysis causes high tension, as in iridectomy for glaucoma a piece of the iris as broad as possible is removed. Nor can paralysis of the ciliary muscle through tearing be held responsible, as paralysis of the oculomotor nerve is not accompanied by glaucoma. The vascular lesions cannot be held responsible, because the collateral circulation of the eye is good and because vascular lesions alone do not greatly influence the circulation (Dietrich). Moreover, no thrombi were observed in the ciliary vessels and vortex veins.

Dislocation of the lens is often accompanied by hypertension, so that glaucoma in such cases is usually ascribed to dislocation of the

lens (Thiel, in Schieck and Brückner's "Kurzes Handbuch der Ophthalmologie"). It is true that glaucoma with traumatic dislocation of the lens as a rule has an unfavorable course. Hegner, for instance, found glaucoma in eighteen of twenty-two instances of traumatic subluxation of the lens collected from the clinic at Jena. In every case the result of treatment was unsatisfactory. Nevertheless, dislocation of the lens is not accompanied by glaucoma in all cases, as Hegner found only eighteen cases of glaucoma in a total of forty-eight cases of traumatic dislocation of the lens collected from the clinic at Breslau. Among eleven cases of complete dislocation this author found glaucoma in only two, while among fifteen cases of dislocation into the anterior chamber (in eight cases traumatic and in seven spontaneous) glaucoma occurred in fourteen. Consequently, glaucoma supervenes only in the majority of cases of dislocations into the anterior chamber of the eye.

It has already been pointed out that in the cases reported in the literature the percentage of dislocations of the lens is greater in cases in which the course is unfavorable, but also that the percentage of other serious intra-ocular lesions is higher. This fact has issued from a comparison of all the instances collected from the literature, and it suggests that dislocation of the lens must be regarded simply as a symptom of serious damage to the eye. This is well illustrated by the two globes the microscopic features of which I have described. Moreover, it is well known that congenital dislocation of the lens may exist quietly for a long time. The fact that glaucoma sometimes manifests itself in these cases cannot be applied to normal eyes. It is also known that a dislocated lens may lie in the vitreous for years without causing any disturbance. Dislocation of the lens alone, therefore, is no direct cause for glaucoma. This is best illustrated by cases of dislocation of the lens in which glaucoma occurs if the patient lies prone and in which the tension returns to normal as soon as the patient lies on his back (Beccaria, 1893; Sabata, 1931). Even dislocation of the lens into the anterior chamber without glaucoma has been described (König, 1895). Finally, traumatic glaucoma is seen so often without dislocation of the lens that one may safely conclude that dislocation of the lens (excepting anterior dislocation) plays no leading part in causing traumatic glaucoma. In one of my own cases (case 12) dislocation of the lens was even accompanied by hypotony.

Regarding the relation between cataract and traumatic glaucoma, it may suffice to state that in the vast majority of cases of traumatic glaucoma no cataract was noted.

Finally, lesions of the nervous system remain to be discussed.

Clinically, the influence of lesions of the nervous system is demonstrated by the occurrence of glaucoma with herpes zoster.

Experimentally, also, there is enough evidence to demonstrate this nervous influence. In experiments on contusion in rabbits Leplat and Schmidt and de Decker noticed that the tension of the sound eye varied with the tension of the other eye, although to a lesser degree. Leplat also noticed, following hypertony, a rise of the albumin-content of the aqueous in both the sound eye and the injured eye. Magitot found that after placing a weight on one eye the tension rises above normal in both eyes. In addition, axon reflexes mediated by the trigeminal nerve are known to exert a powerful influence on the intra-ocular pressure (Duke-Elder) while the sympathetic nervous system also plays an important part.

In traumatic glaucoma the presence of lesions in the nervous system was deduced from the presence of corneal anesthesia by Garnye. Their presence must also be surmised in cases of traumatic glaucoma without visible signs of injury (Tillema, seventh case; Magitot, 1917, second case; Brand, 1905). The patients in the latter cases may have been predisposed, but I consider this unlikely in my case. In this case glaucoma was followed by hypotony. This is a reason for thinking that it represents a transitional stage between simple contusion and traumatic glaucoma. It will be remembered that many of the patients with simple contusion exhibited no lesions, although instability of tension was present in every instance. In such cases taken at random predisposition is highly improbable.

If the variations of pressure after contusion are caused by neurovascular reactions (Leplat, Schmidt and de Becker and others), it is reasonable to suppose that traumatic glaucoma is also based on such reactions. However; while simple hypertony and hypotony may be regarded as the reaction of the normal mechanism to a blow, traumatic glaucoma must be looked on as a pathologic reaction. In the former the healthy neurovascular system readjusts itself; in the latter it is hampered by lesions in the nervous system such as I have demonstrated microscopically.

PROGNOSIS

Previously I have drawn attention to the fact that in cases in which the course is unfavorable more serious lesions of the inner part of the eye are present. From this one may conclude that the presence of such serious lesions suggests a doubtful prognosis. Especially, dislocation of the lens and hemorrhage into the vitreous have proved to be bad signs.

TREATMENT

The cases reported by Magitot and by myself prove that in cases of simple hypertony and hypotony the condition readjusts itself without any treatment. It is sufficient to apply a protective bandage. As the

real cause is uncertain and as the effect of drugs is not clear, even in animals (Leplat's experiments), it is better to refrain from interfering. As glaucoma may manifest itself after every contusion, it is inadvisable to use mydriatics.

Although it was difficult to determine the influence of atropine or scopolamine, it is worthy of note that in the cases reported by Marx and Quatero, Myers, Sala (second case) and Cantonnet the mydriatic was replaced by miotics as soon as the tension rose and that in those cases visual acuity remained unimpaired. It gives one food for thought, however, that in cases reported by Peters (second case), Scheffels, Jacobson (first case), Brand, Hirschberg and Cordes and Horner (first and third cases) and, probably, also in one of my own cases, atropine was administered when the tension was high and that in those cases visual acuity was reduced in every instance. In one of Fromaget's cases atropine had been discontinued eight days before the onset of glaucoma, and yet visual acuity was reduced 1/10. In this case rupture of the sclera was present, which indicates that the eye had suffered a severe contusion and that the disease would have had an unfavorable course in any case.

One may conclude, therefore, that in a number of cases the course has been favorable with the cautious administration of atropine. Yet it seems inadvisable to use a powerful mydriatic, for one would not think of prescribing a mydriatic for patients predisposed to glaucoma. Others also have warned against the use of mydriatics in cases of contusion (Wagenmann; Hardesty, 1931). Green observed a marked rise of tension after the administration of atropine, while Luedde observed a similar effect of eucatropine. Moreover, the indication for the administration of mydriatics is not clearly defined. Some authors use it to obtain quick resorption of a hyphemia or to prevent the onset of "traumatic iritis." Hyphemias are generally resorbed quickly and spontaneously, while the occurrence of so-called traumatic iritis without a perforating wound is exceptional. Others administer a mydriatic to counteract hypotony. In the literature I have found no mention of cases of hypotony in which the course was unfavorable. This, then, need not be an indication. It is much more important to prevent glaucoma, the course of which is always uncertain, than to cure hypotony.

It is also advisable to warn patients against exertion for one week, as Cordes and Horner have observed severe hemorrhage with serious damage to vision.

Experience has shown that in many cases an attack of acute glaucoma is favorably influenced by pilocarpine and physostigmine. If miotics do not suffice one might consider paracentesis of the anterior chamber, because many observers have had good results. In older

patients and in cases in which paracentesis does not give quick relief it is better to carry out iridectomy, for two reasons: first, because experience has shown that this operation is generally efficacious in nontraumatic acute glaucoma and, second, because it is impossible to exclude predisposition for glaucoma in elderly patients.

It is difficult to outline a definite course of treatment for the other forms in which traumatic glaucoma manifests itself. The instances collected from the literature cannot teach one much here, because the cases in which the result of treatment was unfavorable are also those cases in which injury was most serious. At most, one can advise to try the operations for glaucoma in turn.

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DARK ADAPTATION AS A CLINICAL TEST

FURTHER STUDIES

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The study of dark adaptation is a study of a phase of retinal function, and if one accepts the von Kries theory it is specifically an evaluation of rod efficiency. In a previous paper I reported the results of the tests of dark adaptation obtained in a study of 1,008 eyes¹ and in another paper I presented a chart on which such findings may be plotted to enable one to compare the results more easily.²

Almost simultaneously with the beginning of dark adaptation there is a regeneration of the visual purple. Either some failure of this regeneration or a disease of the deeper structure of the retina or choroid may be the principal cause of pathologic dark adaptation. That the retina is richly supplied with vitamin A was shown by Yudkin and his collaborators³ in experiments on animals in which healthy retinal tissue was fed to rats deprived of vitamin A in the diet, with improvement of their symptoms. Wolff⁴ and Moore,⁵ in examining sections of liver tissue chemically by the antimony trichloride technic of Carr and Price, using a Lovibond tintometer, showed the liver to contain the main reserve of vitamin A in the body.

A review is given in this paper of a study of 240 eyes (120 patients were examined); 229 were free from actual retinal disease. Vision, with the few exceptions stated later, was normal or nearly so. I divided into three groups the 120 patients examined. Each of these groups showed factors which might affect the nature of the graph for dark adaptation.

Group 1 included patients already described in my previous paper¹ but examination of whom gave further confirmation of the previously

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1. Feldman, J. B.: Dark Adaptation as a Clinical Test, *Arch. Ophth.* **15**:1004 (June) 1936.

2. Feldman, J. B.: A Graph for Recording Results in Dark Adaptation, *Am. J. Ophth.* **19**:510, 1936.

3. Yudkin, A. M.; Kriss, M., and Smith, A. H.: Vitamin A Potency of Retinal Tissue, *Am. J. Physiol.* **97**:611, 1931.

4. Wolff, L. K.: On the Quantity of Vitamin A Present in the Human Liver, *Lancet* **2**:617, 1932.

5. Moore, T.: Vitamin A Reserves of the Human Liver in Health and Disease, *Lancet* **2**:669, 1932.

found data. This group also included patients who were tested with various colors for light adaptation previous to the dark adaptation test. Ordinarily, white light is used in adapting the eyes of every patient to light before the studies of dark adaptation are made. There were also in this group a few patients with poor vision due to such conditions as amblyopia and conical cornea; patients with choroiditis, glaucoma or retinal detachment, and several patients with miscellaneous diseases.

Group 2 was composed of patients with a disease of the blood or with a pathologic condition of the blood vessels (arteriosclerosis). Since it is through the circulation that nutriment is brought to retinal tissue, it was thought worthy of study to determine whether a pathologic condition of the blood or the blood vessels might aid in disclosing a common factor as a cause for the pathologic graph.

Group 3 consisted of patients with diseases which have been found to be related to vitamin A deficiency. The lack of vitamin A might affect the results of the dark adaptation study. This group included patients with avitaminosis A, metabolic disturbance, calculi of the genito-urinary tract, diabetes mellitus or disease of the liver or gall-bladder and correlated diseases, and pregnant and sterile patients.

METHOD OF STUDY

All the patients were studied in the usual manner.¹ The cases were unselected. When possible, a study of the blood was carried out. I have noticed that the only significant parts of the routine complete blood count seemed to be the percentage of lymphocytes and the color index, as related to the dark adaptation study; therefore, in the tables which follow I have incorporated only these two findings. In many cases it was impossible for one reason or another to obtain even these findings for purposes of analysis. In cases in which only one eye showed a pathologic condition in dark adaptation this is designated by "pathologic $\frac{1}{2}$." "Pathologic +" and "pathologic ++" signify varying degrees of marked pathologic change in the dark adaptation graph. Focal infection was eliminated as far as possible in all cases. All the patients examined were adults, except for 5 diabetic patients (from 6 to 16 years old).⁶ That age, per se, does not affect dark adaptation has been noted previously.¹

Preexposure of Eyes to Light Other than White.—In all cases in which studies of dark adaptation are to be made the patient must look at a pure white light for a definite period. This is called light adaptation. In my cases this period lasted from three to five minutes.

6. These were from the clinic for patients with diabetes of St. Christopher's Hospital for Children.

Krause⁷ quoted Kuhne, Boll and Weiner as noting a qualitative alteration of the visual purple when it is exposed to light of different colors. Red light changes the visual purple to a brownish color; blue light, to rose, and yellow light, to yellow. Two patients who had been previously tested and whose eyes had been checked as normal were used in this experiment. The preexposure to light in each case was three minutes, and in each test a different color was placed before the patient. As in my routine studies, each sitting in the test of dark adaptation was thirty minutes, and readings were taken every three minutes. Preexposure to yellow and red light delayed dark adaptation in both patients greatly beyond the thirty minute period. Only the preexposure to blue light did not materially affect the results of the dark adaptation studies to a great extent.

A review of the cases studied which were obtainable to date are presented.

STUDIES ON GROUP 1

Amblyopia.—Visual defects of various degrees do not usually affect dark adaptation to a serious extent. Surprisingly, the patient with dark adaptation marked pathologic $\frac{1}{2}$ and 45 per cent lymphocytes (table 1) showed a normal dark graph for the amblyopic eye and a pathologic dark adaptation graph for the eye with normal vision.

Conical Cornea.—In my last study of dark adaptation I did not have any patients with conical cornea, although I felt at that time that visual error did not affect the dark adaptation.¹ Two patients were examined in this study. The vision of one was 3/60 for both eyes, with a concomitant convergent squint of 30 degrees. The other patient had vision of 6/60 in one eye and 6/12 in the other. Both patients showed a high normal dark adaptation reading. No studies of the blood could be made.

Choroiditis.—The two cases the data for which are given in table 2 are of great interest. In the first case the choroiditis was confined only to the macula of each eye, and yet the dark adaptation graph was pathologic, despite the fact that the macula is concerned only with light adaptation. In the second case a small patch of choroiditis was noted, but the important fact was the positive diagnosis of glaucoma associated with the normal graph for dark adaptation. In a case of this sort one would expect to find a pathologic graph.

Glaucoma.—These studies are based on reports of cases given in a previous article.¹ However, reports of studies of the blood were not included in that publication. All the patients were glaucomatous and had pathologic dark adaptation of various degrees. The two cases in

7. Krause, A. C.: *The Biochemistry of the Eye*, Baltimore, The Johns Hopkins Press, 1934.

which the dark adaptation was marked pathologic $\frac{1}{2}$ merit a brief review. A. L. R., aged 51, had a condition diagnosed as glaucoma of both eyes. The tension at time of examination was 26 mm. in the right eye and 20 mm. in the left. Dark adaptation was normal in the right eye and pathologic in the left. The other patient, E. G., had a condition diagnosed as chronic simple glaucoma of both eyes; the right eye was blind. However, the left eye showed normal dark adaptation at this

TABLE 1.—*Data for Patients with Amblyopia **

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Epilepsy in 1 case.....	Normal	X	X
1	Epilepsy	Pathologic $\frac{1}{2}$	45	-1
1	Pathologic $\frac{1}{2}$	X	X
1	Pathologic	52	-1

* In this and the following tables X means that this test could not be performed.

TABLE 2.—*Data for Patients with Choroiditis*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	Macular choroiditis	Pathologic	49	X
1	Small patch of tuberculous choroiditis; juvenile glaucoma; tension 50; field now normal, with tension 18..	Normal	36	-1

TABLE 3.—*Data for Patients with Glaucoma*

Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Pathologic $\frac{1}{2}$	45; 39	1
4	Pathologic +.....	26 (average)	-1
5	Pathologic ++.....	49; 44; 36; 50; 35	-1
1	Pathologic.....	41	1

sitting, despite the fact that the tension was 30 mm. (Schiötz) at the time. A résumé of the cases is given in table 3.

Miscellaneous Diseases.—An interesting group of 8 cases fall under this classification. A patient with a provisional diagnosis of myxedema showed definite signs of glaucoma, with a tension of 31 mm. and a contracted field. This patient had a normal dark adaptation graph. Another patient who had a dislocated lens and tension of 35 mm. also showed a normal graph. The patient with keratoconus associated with opacities of the vitreous in the left eye and with vision of 6/60 showed a normal graph for that eye. Three patients were suspected of having angioneurotic edema. All these showed a pathologic graph for dark adaptation.

Retinal Detachment.—This is a common cause of faulty regeneration of the visual purple (pathologic dark adaptation). Krause⁸ quoted Andogsky as stating that he observed no regeneration of the visual purple after detachment of the retina of a rabbit.

STUDIES ON GROUP 2

This group, as has been mentioned, consisted of patients with diseases of the blood or the blood vessels. It is unfortunate that this group did not contain more outstanding examples of pathologic changes and

TABLE 4.—*Data for Patients with Retinal Detachment*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	High myopia	Pathologic +	X	X
1	Vitreous opacity	Pathologic ++	42	1+

TABLE 5.—*Data for Patients with Miscellaneous Diseases*

Provisional Diagnosis	Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
Beginning myxedema	1	Ocular hypertension; tension 31 mm. [Sehiötz]); contracted field	Normal	29	1
Dislocated lens of left eye	1	Tension 35 (Sehiötz)	Normal	X	X
Hypertension (arterial)	1	Blood pressure 210/130	Pathologic ½	X	X
Keratoconus and vitreous opacities of left eye	1	Vision 6/6, 6/60	Normal	53	-1
Chronic arthritis	1	Three infected teeth; low grade ethmoiditis	Normal	37	1
Angioneurotic edema (?)	3	Eosinophils 4 per cent in each case	Pathologic	32; X; 44	-1; X; 1+

that, for instance, a patient with 9 per cent eosinophils should be regarded as showing evidence of disease. A comparison between this patient and the patient with trichinosis with 30 per cent eosinophils is enlightening. The latter patient had no other illnesses and was improving from his infestation. He had a normal dark adaptation graph. It would seem from this that it was not the eosinophilia but the underlying pathologic condition which really affected the results of the dark adaptation study. So also in the other blood diseases it appeared that when the condition was active the graph showed evidence of a moderately pathologic condition, whereas in the cases in which the patient was on the road to recovery the graph was normal or nearly so.

8. Krause,⁷ p. 82.

Of 5 patients showing a rise in the percentage of eosinophils 1 had postoperative uveitis; vision in each eye was perception of movements of the hand. This patient had active involvement, with 15 per cent eosinophils. The dark adaptation graph showed evidence of marked pathologic changes.

There were 2 cases of chorioretinitis. One patient had 8 per cent eosinophils and dark adaptation that was classed as pathologic ++. The other had 9 per cent eosinophils; the disease was not as severe

TABLE 6.—*Data for Patients with Diseases of the Blood*

Diagnosis	Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
Secondary anemia...	2	Pathologic ½	23; 14	1+; -1
Pernicious anemia...	2	Pathologic	48; 22	1+; 1+
Pernicious anemia...	1	Normal	22	1+
Secondary anemia...	1	Normal	20	1+
Lymphatic leukemia	1	Pathologic	95	1+
Hodgkin's disease and syphilis	1	Interstitial keratitis; Wassermann reaction 3+	Pathologic ½	51	-1

TABLE 7.—*Data for Patients with Arteriosclerosis*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
2	Hemorrhagic retinitis	Pathologic +	X	X
3	Normal	X	X
1	Normal	42	-1
1	Pathologic ½	X	X
1	Hypertension	Pathologic	33	-1
1	Hypertension	Pathologic +	X	X
1	Hypertension	Pathologic	35	-1
1	Pathologic +	23	-1
1	Pathologic	33	1+
2	Chronic nephritis; secondary anemia	Pathologic	19; 42	-1, -1
1	Secondary anemia	Pathologic +	23	-1
1	Hypertension	Pathologic +	X	X

as in the first case, and the graph did not show evidence of as pathologic a condition. Further observations might well be made in subsequent cases on the presence of eosinophilia in association with chorioretinitis.

There was 1 patient with marked arteriosclerosis and hypertension who had 9 per cent eosinophils and dark adaptation that was classed as pathologic ++. This patient was 71 years of age.

Arteriosclerosis.—In a previous review¹ it was pointed out that arteriosclerosis gave pathologic dark adaptation readings for all but 4 of the 31 patients examined.

The group now reported on consisted of 16 additional patients. The results were somewhat similar to those in the former study, as 12 of these patients showed a pathologic graph.

STUDIES ON GROUP 3

This group consisted of patients with such diseases as are supposed to be associated with vitamin A deficiency.

Vitamin A is colorless, belongs to the alcohol group and has the formula $C_{20}H_{29}OH$ and an absorption band at 324 angstroms (the ultra-violet area). The liver contains the main reserve of vitamin A in the body; vitamin A is also found to a great extent in the retina.⁹

Mathews¹⁰ quoted Wald as accounting for the presence of vitamin A in the retina as follows:

1. Vitamin A is extracted from the blood by the retina, more by the pigment layer than the rods, and combines with protein to make visual purple.

2. When visual purple is exposed to light it becomes visual yellow (a photochemical change). The visual yellow then breaks up (a chemical change) into vitamin A and colorless products. The process is then repeated, vitamin A combining with protein to form visual purple. What little vitamin A is lost in the process is replenished from the blood.

Tansley¹¹ has shown by photographing various retinas the quantitative difference in regeneration of the visual purple in normal rats and rats with vitamin A deficiency. It was previously noted that a delay in regeneration of the visual purple frequently caused dark adaptation to become pathologic.

Eusterman and Wilbur¹² claimed frequent causes of vitamin A deficiency to be: (a) the growing demand of the tissues of the body of the child; (b) hepatic and intestinal disease in adults with poor absorption and storage; (c) an increased demand for vitamins, as in disease or infections. Xerophthalmia is the most common sign of vitamin A deficiency, but it is not necessarily a constant evidence of the presence of the condition. Moore⁵ cited the cases of 12 patients who at autopsy showed no reserve of vitamin A and yet during life presented no evidence of xerophthalmia. Can a person who has enough vitamin A in the system suffer from avitaminosis? Mathews¹³ expressed the belief that this is possible. He stated: "If the vitamin, which is an alcohol, esterifies with phosphoric acid before it can become active, and

9. Foster, J.: Vitamins in Ophthalmology, Arch. Ophth. **15**:1160 (June) 1936.

10. Mathews, A. P.: Principles of Biochemistry, Baltimore, William Wood & Company, 1936, p. 412.

11. Tansley, Katharine: The Regeneration of Visual Purple: Its Relation to Dark Adaptation and Night Blindness, J. Physiol. **71**:442, 1931.

12. Eusterman, G. B., and Wilbur, D. L.: Chemical Features of Vitamin A Deficiency, J. A. M. A. **98**:2054 (June 11) 1932.

13. Mathews,¹⁰ p. 414.

if a protein component is also necessary for its activity, it might be possible by the action of a suitable phosphatase, acting on such a union to produce the symptoms of avitaminosis even when Vitamin A was present as such in the tissues."

The data for the patients examined in group 3 are given in the following sections.

Atrophic Rhinitis.—Cody¹⁴ claimed that vitamin A is necessary to the nutrition of nasal, tracheal and aural epithelium. He quoted Glassheib and Fleishman as giving the pros and cons relative to the etiologic importance of vitamin A in atrophic rhinitis. Only 1 case of this type

TABLE 8.—Data for Patients with Avitaminosis A

Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	Pathologic.....	37	X
1	Pathologic.....	X	X
1	Pathologic.....	33	X
1	Pathologic.....	37	—1

TABLE 9.—Data for Patients with Metabolic Disturbances

Number of Cases	Basal Metabolic Rate	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	+17	Cystic goiter.....	Pathologic ½	X	X
1	+16	Anemia.....	Pathologic ½	50	1+
1	+17	Cystic goiter.....	Pathologic	39	1
1	—37	Myxedema.....	Pathologic	30	—1
1	—11	Prostatitis.....	Pathologic	37	—1

was observed; there was pathologic ½ dark adaptation. No tests of the blood could be carried out.

Avitaminosis A.—There were 4 cases in which vitamin A deficiency was suspected. In all the dark adaptation graph was pathologic.

Disturbances of the Basal Metabolism.—Few patients with metabolic disturbances could be obtained for study. Of the 6 patients whose basal metabolic rate was determined, only 1 had a definitely abnormal rate. This was a patient with myxedema. The reading was —37. In this case the dark adaptation graph was pathologic. The other patients in this group showed little pathologic change in dark adaptation. It will be noted that 1 patient with a normal reading (—11) for the basal metabolism also showed a pathologic graph, but this graph was not as pathologic as that of the patient with myxedema. In this particular case

14. Cody, C. C.: Relation of Vitamin A, D, B and G to Otolaryngology, Arch. Otolaryng. 16:664 (Nov.) 1932.

there was definite prostatitis, which might account for the pathologic reading.

Calculi of the Genito-Urinary System.—Wolbach and Howe,¹⁵ Higgins¹⁶ and many others have found in avitaminosis A in the rat metaplasia of the tissue of the urinary tract and pelvis of the kidney, which resembled to a great extent the finding for a child who died of avitaminosis. This case was reported by Wilson and DuBois.¹⁷ In the rat, however, in addition to the metaplasia due to avitaminosis A, stones usually form in the bladder or the kidney. McCarrison and Ranganathan¹⁸ noted the importance of the rôle of vitamin A in the formation of calculi by saying that the phosphorus and calcium balance will not entirely prevent the formation of calculi unless a normal quantity of vitamin A is present.

McCarrison¹⁹ expressed the belief that the metaplasia of the tissue of the urinary tract caused by avitaminosis A may form a nidus around which a deposit of calculus may form. That avitaminosis A is not the sole cause of calculi has been contended by Bliss, Livermore and Prather,²⁰ who found that from 38 to 43 per cent of rats did not show calculi when on a diet deficient in vitamin A. Higgins²¹ found by laboratory experiments that calculi dissolved in rats who were fed on a vitamin A diet.

All these experiments were performed on animals. The Council of Pharmacy and Chemistry of the American Medical Association²² in a review of the subject of vitamins found "inadequate evidence to warrant the claim that ingestion of vitamin A will prevent calculi in man."

The association between vitamin A and the visual purple was noted previously in this paper. It was also stated that faulty regeneration

15. Wolbach, S. B., and Howe, P. R.: Tissue Changes Following Deprivation of Fat-Soluble A Vitamin, *J. Exper. Med* **42**:773, 1925.

16. Higgins, C. C.: The Experimental Production of Urinary Calculi, *Urol. & Cutan. Rev.* **38**:33, 1934.

17. Wilson, J. R., and DuBois, R. O.: Report of a Fatal Case of Keratomalacia in an Infant with Postmortem Examination, *Am. J. Dis. Child.* **26**:431 (Nov.) 1923.

18. McCarrison, R., and Ranganathan, S.: Researches on Stones: XII, *Indian J. M. Research* **19**:55, 1931.

19. McCarrison, R.: The Causation of Stones in India, *Brit. M. J.* **1**:1009, 1931.

20. Bliss, A. R.; Livermore, G. R., and Prather, G. O.: The Relation of Vitamin A and D to Urinary Calculus Formation, *J. Urol.* **30**:639, 1933.

21. Higgins, C. C.: Production and Solution of Urinary Calculi, *J. A. M. A.* **104**:1296 (April 13) 1935.

22. The Status of Certain Questions Concerning Vitamins, report of the Council on Pharmacy and Chemistry, *J. A. M. A.* **106**:1733 (May 16) 1936.

of the visual purple is caused by avitaminosis A. The phenomena in this faulty regeneration are instrumental in producing pathologic dark adaptation. I next attempted to ascertain the possible relationship between dark adaptation, renal calculi and avitaminosis A. Dr. William Ezickson²³ permitted me to examine 25 of his patients with calculi of the renal tract in this study. In addition to these, 3 other patients were examined who were obtained from other sources. The diagnosis in all the cases in this group was verified by roentgen, cystoscopic and other examinations. Two patients of this group had just passed stones. They were found to show no abnormality on roentgen examination, but one showed pathologic dark adaptation, and the other, pathologic $\frac{1}{2}$.

Six months after the roentgen study that showed no abnormal condition in the patient with the pathologic graph, a return of symptoms made advisable a second roentgen study. This study gave evidence of recurrence of stones in the kidney. Unfortunately, I was not able to repeat the dark adaptation study.

For the remaining 26 patients in this group of patients with calculi of the renal tract definite results were obtained with the dark adaptation test. Two patients showed a pathologic $\frac{1}{2}$ curve for dark adaptation. Twenty-three showed a pathologic graph. In the 1 remaining case an erroneous diagnosis had been made. There was no pathologic condition of the renal tract. The graph was normal. The color index in this case was -1 , and the lymphocyte count was 26 per cent.

Of the patients with calculi, 15 showed a lymphocyte count of over 35 per cent. In 18 cases the color index was over 1. Although every patient with calculi in this series showed a pathologic curve, no definite degree of disease was disclosed. The number, size and composition of the calculi bore no discernible relation to the dark adaptation graph. In addition to the patient for whom the erroneous diagnosis of renal disease had been made a number of normal patients with normal dark adaptation graphs were examined, who acted as controls for the patients with calculi.

Diabetes Mellitus.—With the exception of 1 adult, all the patients examined were children whose ages ranged between 6 and 16 years. The graphs obtained in these cases varied from normal to mildly pathologic. Eight patients in all were examined. It is possible that the vegetables, eggs and butter included in a well balanced diabetic diet would tend to limit evidences of avitaminosis A in diabetes.

Diseases of the Liver and Gallbladder and Correlated Diseases.—Krause⁷ quoted Bass and Koyanagi as associating diseases of the liver

23. A complete report of these cases will be published by Dr. Ezickson in a paper in the near future.

with a deficiency in regeneration of the visual purple and night blindness. I was fortunate in being able to study 2 patients with syphilis of the liver. Ordinarily syphilis does not affect dark adaptation. It is interesting to note that Wolff⁴ found a normal amount of vitamin A

TABLE 10.—*Data for Patients with Diabetes Mellitus*

Number of Cases	Comment	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index
1	Pathologie	45	—1
4	Normal	X	X
1	Coma on day before dark adaptation test was carried out.....	Pathologie	X	X
1	Pathologie	X	X
1	Pathologie ½	X	X

TABLE 11.—*Data for Patients with Diseases of the Liver and Correlated Diseases*

Diagnosis	Number of Cases	Results of Dark Adaptation Test	Percentage of Lymphocytes	Color Index	Icterus Index
Ulcerative colitis.....	1	Pathologie ½	35	—1
Chronic nephritis.....	1	Pathologie	38	X	15.0
Cholecystitis.....	1	Normal	X	X
Chronic hepatitis and duodenitis	1	Normal	36	X	8.8
Chronic cholangitis and hepatitis	1	Pathologie	26	X	...
Cholelithiasis; chronic cholecystitis; syphilis	1	Mildly pathologie	30	X	8.8
Chronic hepatitis.....	1	Normal	35	X	8.8
Syphilis of liver.....	1	Normal	32	X	...
Syphilis of liver.....	1	Pathologie ++	X	X	Marked jaundice

TABLE 12.—*Data for Pregnant Patients*

Number of Cases	Results of Dark Adaptation Test	Comment
4	Normal	
1	Pathologie	High myopia
1	Pathologie ½	

TABLE 13.—*Data for Sterile Patients*

Number of Cases	Results of Dark Adaptation Test
4	Normal
1	Pathologie

in syphilitic livers. One of the 2 patients in my series showed a normal graph, which was not unexpected. The second patient, in whom the disease was complicated by marked jaundice, showed a pathologic graph. Jaundice has been known to affect the graph in this manner.

Four nonsyphilitic patients with hepatitis were also studied; of these, 2 had a pathologic graph, and 2 had normal readings.

Pregnancy.—That vitamin A, together with vitamin E, is a factor in fertility and the processes of reproduction is conceded, and was mentioned by Cobb.²⁴ The 1 patient the data for whom are given in table 12 who had pathologic dark adaptation had myopia requiring a —12.00 sphere. Some investigators of dark adaptation have given high myopia as a cause of pathologic dark adaptation. Of the 6 patients examined in my series, 4 showed normal readings. All the patients were about six months pregnant.

Sterility.—Wilson and DuBois¹⁷ found in the patient with sterility whom they examined post mortem a change in the columnar epithelium in the uterine mucosa to the stratified keratinized type. This change would, it is believed, explain the cause of some cases of sterility in which avitaminosis A was a factor. Five patients were examined in my series. Only 1 of these showed a pathologic dark adaptation graph. It was later found that tubo-ovarian disease was the underlying cause for the sterility in this patient.

SUMMARY AND CONCLUSIONS

A review of the results of dark adaptation tests on 120 patients (240 eyes) is reported. A pathologic dark adaptation graph may be due either to failure of regeneration of the visual purple or to a disease of the deeper structure of the retina or choroid. The cases were arbitrarily divided into three groups, according to the factor which was considered as affecting the dark adaptation graph. The groups are:

1. The group of patients with various diseases of the eye.
2. The group of patients with diseases of the blood or blood vessels, which included patients with anemia, patients with eosinophilia and patients with arteriosclerosis.
3. That group of patents in whom avitaminosis A was considered a factor in one or another of several diseases, e. g., renal calculi and metabolic disturbances.

An attempt was made to note the frequency with which pathologic dark adaptation was associated with an increased percentage of lymphocytes in the blood as well as with an increased color index.

An evaluation of different-colored lights for preexposure of the eye (light adaptation) showed that white light is the best and that blue light is next best. Dark adaptation was delayed with preexposure to red or yellow light in the normal eye.

24. Cobb, Percy: Light Sense, in Berens, C.: *The Eye and Its Diseases*, Philadelphia, W. B. Saunders Company, 1936, chap. 19, p. 206.

In cases of group 1 the following were noted:

1. In patients with defective vision due to such conditions as amblyopia or conical cornea the tendency is toward a normal dark adaptation graph.

2. Pathologic graphs are often obtained in cases of choroiditis and a great majority of the cases of glaucoma, chorioretinitis and retinal detachment.

In the cases of group 2 the results of the studies seem to indicate the following:

1. The basic cause of the blood condition was a material factor in determining the dark adaptation graph.

2. Arteriosclerosis in a great majority of cases gave a pathologic graph.

In group 3 the following were noted:

1. The patients with avitaminosis A all showed a pathologic graph.

2. Patients with metabolic disturbances tended to have pathologic readings.

3. Patients with calculi of the genito-urinary tract showed definite pathologic dark adaptation.

4. In diabetes mellitus no consistent results were obtained.

5. In diseases of the liver or gallbladder and correlated diseases the results were inconclusive.

6. Syphilis itself does not affect the dark adaptation graph. However, in the presence of marked jaundice it appears as though this would not apply.

7. Pregnancy and sterility do not materially affect dark adaptation. Examination of the blood was possible in only 75 of the 120 cases studied. In 45 of these 75 cases there was both a pathologic dark adaptation graph and a lymphocyte count of over 35 per cent. Only 5 patients with a normal reading for dark adaptation had a lymphocyte count of over 35 per cent. Two of these had a color index of over 1.

The triad of (a) a high lymphocyte count, (b) a color index of over 1 and (c) a pathologic dark adaptation graph was definitely observed in those patients with renal calculi. Fifteen of the patients showed a lymphocyte count of over 44 per cent. Eighteen showed a color index of more than 1.

The many conflicting results obtained in regard to pathologic dark adaptation are of great interest, and the subject merits further study, particularly in view of the fact that faulty dark adaptation is synonymous

with hemeralopia (night blindness). Attention has been called²⁵ to the many accidents which occur in airplane and motor service in both military and civil life which may be attributable to pathologic dark adaptation. With the foregoing thoughts in mind I shall attempt further study in this subject.

Drs. H. M. Eberhard, William Ezickson, J. D. Paul and O. J. Toland permitted me to observe a few of the cases here reviewed.

25. Adams, Dorothy: Report of Committee on Physiology of Vision: II. Dark Adaptation, Medical Research Council, Special Report Series, no. 127, London, His Majesty's Stationery Office, 1929. Percival, A. S.: The Light Sense, Tr. Ophth. Soc. U. Kingdom **40**:311, 1920. Park, I. O.: Preliminary Observations on Vitamin A Deficiency as Shown by Studies with Visual Photometer, J. Oklahoma M. A. **28**:357, 1935.

SPASM OF THE CENTRAL RETINAL ARTERY IN RAYNAUD'S DISEASE

REPORT OF A CASE

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In reviewing the literature on Raynaud's disease, it is found that ocular complications occur infrequently and spasm of the central artery of the retina is exceedingly rare. In fact, Raynaud's disease itself is not common.

Raynaud's disease was first described as a distinct malady in 1862 by Maurice Raynaud.¹ In describing Raynaud's disease, DaCosta² said that it is usually regarded as a vasomotor neurosis, and is seen most frequently in children and young women but sometimes in men. The etiology is not known. Cecil and Kennedy³ said that the disease passes through three stages: local syncope, local asphyxia and local gangrene. Local syncope is characterized by vasoconstriction of the affected parts which makes them pale and cold. The fingers and toes are the parts usually involved, but the disease may affect the ears, nose, lips, chin and nates. There is a feeling of deadness, usually accompanied by severe pain or paresthesia of the parts. This stage is followed by local asphyxia or cyanosis, of a toe or finger usually. Local asphyxia may persist for weeks or months before gangrene starts. The parts are usually symmetrically affected, and attacks of Raynaud's disease may recur several times without ending in gangrene. The disease is seldom fatal.

It seems possible for spasm of the retinal vessels to occur in Raynaud's disease, since hemiplegia has been reported in several cases of this disease, which was probably due to spasm of the cerebral vessels.

1. Raynaud, Maurice: *De l'asphyxie locale et de la gangrène symétrique des extrémités*, Paris, Rignoux, 1862.

2. DaCosta, J. C.: *Modern Surgery, General and Operative*, ed. 9, Philadelphia, W. B. Saunders Company, 1925, p. 103.

3. Cecil, R. L., and Kennedy, Foster: *Textbook of Medicine*, Philadelphia, W. B. Saunders Company, 1930, p. 1427.

Foster Moore⁴ examined a number of patients specifically for spasm of the retinal vessels, but in no case was there any evidence of its presence at the time of examination.

Shinkle,⁵ in 1924, reported a case of Raynaud's disease of the feet in a woman aged 44 years. Her left eye suddenly became blind, though the pupil still reacted to light. Her eye was examined by Drs. F. H. Lamb and Louis G. Heyn. They found the retinal arteries uniformly shrunken. Under the influence of amyl nitrite the retinal arteries dilated, but they contracted again as soon as the effect of the drug wore off. The patient was given large doses of potassium iodide and hypodermic injections of sodium nitrite. She gradually improved, and vision slowly returned to the left eye, but it never became normal.

E. B. Dunphy,⁶ in a thesis read before the American Ophthalmological Society in 1932, reviewed the literature on the ocular complications of Raynaud's disease and also reported a case in which there was bilateral spasm of the central retinal arteries. The patient was a white girl aged 9 years, for whom the diagnosis at first was apparently rheumatic fever with purpura. After several months of slow improvement, attacks of local syncope, asphyxia and gangrene of the finger-tips occurred symmetrically, and the toes on each foot became cold and painful. The diagnosis then was thought to be Raynaud's disease. Six months after the diagnosis of rheumatic fever the patient complained of poor vision in the right eye. The house officer examined her and reported a "definite red papule lateral to the disk, with the rest of the retina pale." Dr. Dunphy examined her six months later, when she could only count fingers at 6 feet (182.8 cm.) with the right eye. At this time definite pallor of the right disk was noted, and the retinal arteries were extremely small. The retina was normal in color, and the red spot in the macula had disappeared. In a few days the left eye suddenly became blind. Dr. Dunphy reported blanching of the whole retina, with a cherry-red spot in the macular region. The arteries were extremely thin. The patient was given inhalations of amyl nitrite; paracentesis of the cornea was done, and she was given spirit of glyceryl trinitrate, but with no effect on the visual acuity or the fundus picture. Later, bilateral lumbar sympathectomy, right cervical sympathectomy and removal of the stellate ganglion were performed. Two years later she could count fingers at 3 feet (91.4 cm.) with the right eye and had the barest perception of light in her left eye.

4. Moore, R. F.: *Medical Ophthalmology*, ed. 2, Philadelphia, P. Blakiston's Son & Company, 1925, p. 95.

5. Shinkle, C. E.: Raynaud's Disease Involving the Feet, the Left Retina and the Heart Wall, *J. A. M. A.* **83**:355-356 (Aug. 2) 1924.

6. Dunphy, E. B.: *Tr. Am. Ophth. Soc.* **30**:420-430, 1932.

Allen and Brown⁷ reported one hundred and forty-seven cases of uncomplicated Raynaud's disease at the Mayo Clinic during the period from January 1920 to July 1931. Of these patients, one had attacks of blindness suggestive of intermittent spasm of the retinal arteries.

Owing to the infrequency of spasm of the central retinal artery in Raynaud's disease, we should like to add the following case to those previously reported.

REPORT OF CASE

History.—K. G. E., a white man aged 39 years, was referred to us by Dr. O. W. Leonard on Oct. 23, 1936, because of sudden blindness of the right eye on October 10, which had persisted. There was no history of trauma or previous disease of the eye, and the blindness was not accompanied by pain. The family history offered nothing of interest other than that the patient was of German-Irish descent. The past history was interesting and of particular importance. The patient had had Raynaud's disease for eleven years, since 1925. He suffered with numbness and cold toes in 1925, accompanied by severe pain. This resulted in gangrene and loss of part of the great toe of the right foot. He had no further trouble until 1930, when he had a similar attack resulting in gangrene of the second and third toes of the right foot; these were amputated completely. He was a radio operator and went about his work with only an occasional attack of coldness, numbness and pain of the fingers and toes until 1935. At this time he had a similar attack, but more severe, which resulted in gangrene and the loss of the greater part of the second and third fingers of the right hand. He has had no gangrene of the affected parts since 1935 but frequently has recurrent attacks of cold fingers and toes, with numbness and pain. The condition has aggravated him to such an extent that he resorts to alcoholic stimulants at least twice a week and probably more often. He has smoked from twenty to thirty cigarettes daily for the last twenty-two years.

Examination of the Eyes.—Vision of the right eye was limited to the barest perception of light in a very small area of the temporal field. Vision of the left eye was 20/20. The right fundus showed a pale, opaque, milky-white retina, especially in the region of the fovea centralis and the optic papilla. There was a bright red spot in the center of the fovea. The retinal arteries were very thin and small, and several branches were bloodless; the retinal veins were apparently normal. The arteries in the left fundus were slightly smaller than the average, but the veins appeared normal. The right fundus of this patient showed a typical textbook picture of embolism of the central artery.

Diagnosis.—There seemed to be no doubt as to the diagnosis of Raynaud's disease, since the patient had been seen by many physicians, who had concurred in this diagnosis. In view of this diagnosis and the history of the patient, our diagnosis was spasm of the central artery rather than an embolus. Broken columns of blood could be seen moving slowly in the arteries away from the disk, more rapidly at some times than at others.

Treatment and Course.—Since this eye had been blind for thirteen days, we realized that although the retinal circulation were restored the vision would not return because the retina would already be atrophic. However, we advised imme-

7. Allen, E. V., and Brown, G. E.: Raynaud's Disease: A Clinical Study of One Hundred and Forty-Seven Cases, J. A. M. A. **99**:1472-1477 (Oct. 29) 1932.

diate hospitalization for possible lumbar and cervical sympathectomy for the Raynaud disease and at the same time to see whether we could restore normal retinal circulation. The patient did not consent, and we did not see him again until December 4.

In the interim he was seen by Dr. E. W. Carpenter,⁸ who will probably give a report of the end-result at a later date. When the patient returned to us on December 4, vision of the right eye was not improved. The retina was normal in color; the red spot in the fovea centralis had disappeared, and the arteries were somewhat larger. More blood was flowing through the arteries, but the smaller branches still appeared thin and bloodless. The disk showed definite pallor, which was more marked on the temporal half. The left fundus was essentially normal. The patient complained of the fact that his toes and fingers became cold and numb; so he was given large doses of potassium iodide. By December 29 the coldness and numbness had disappeared again.

When he was last seen, on Jan. 22, 1937, vision of the right eye was still only the barest perception of light in a small area of the temporal field. The fundus picture was essentially the same as on Dec. 4, 1936. He had not been bothered with coldness and numbness of his toes and fingers for over three weeks and was still taking potassium iodide.

8. Carpenter, E. W.: Report by letter to the author.

THE GLAUCOMA CLINIC OF THE HERMAN KNAPP MEMORIAL EYE HOSPITAL

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For many years ophthalmologists have realized that progress toward the solution of the problem of glaucoma has been so dishearteningly slow that something more definite must be achieved in regard to this question.

The symposium on glaucoma at the Thirteenth International Congress of Ophthalmology, held in Amsterdam, the Netherlands, in 1929, did nothing to improve the situation. Duke-Elder, Magitot, Wessely and von Haagen, who reviewed the subject, confessed that the mystery was as profound as ever.

Having studied the problem for many years, our colleagues and we felt that the prevailing theories were leading nowhere. Gradually one of us developed a theory of his own as to the nature of glaucoma, differing in many respects from prevailing theories. We felt, also, that no progress would be made except by means of a carefully thought out plan of action not only involving theoretical considerations and laboratory investigations but combining these with a well organized, systematic and intensive clinical study of each patient with glaucoma.

With these ideas in mind a special clinic for patients with glaucoma was opened at the Herman Knapp Memorial Eye Hospital in October 1935. A modest sum of money was granted by the trustees of this institution. The staff of the clinic consisted of a director, an assistant and a medical diagnostician. The patients were referred to the clinic by the members of the staff of the Herman Knapp Memorial Eye Hospital. After a careful examination of the eyes each patient was referred to the medical diagnostician, who, in addition to recording the findings of the physical examination, recorded the personal and the family history as completely as possible. With all the available data at hand, the staff of the clinic was better able to see the entire picture and to be guided by it when prescribing treatment. Were space not limited, a number of instances could be cited in which the course of glaucoma was unmistakably influenced by constitutional or endocrinologic aberrations.

The examination of the eyes in each case was done in a leisurely fashion and thoroughly, at intervals varying from once a week to once a month. This, in addition to routine tests, included slit lamp study, tonometric examination and refraction. The peripheral and central fields were checked at least every six weeks. Special emphasis was laid on every detail of the patient's complaints. Nothing that the patient had to say regarding his ocular or general condition was considered insignificant. The fact was not lost sight of that in patients with glaucoma the threshold between the eye and the impulses reaching it from the rest of the organism is lowered; that a cold in the head, a febrile disease, a mental upset, a gastro-intestinal disturbance and other conditions often induce a rise of the ocular tension, sometimes of considerable proportion. Inquiry was made not only into the physical condition of the patient but also into his mental life. Lately, the addition to the staff of a psychiatrist for the sole purpose of studying the patients from the psychiatric point of view has been considered.

The clinic succeeded in securing the services of a full time ophthalmologist, intensely interested in the problem of glaucoma, for the routine examination of the eyes of each patient. This gave the other members of the staff more time to study the entire picture in each case and particularly any interesting or unusual features.

The examination of the ocular tension at regular intervals and at a definite time after the use of miotics furnished a formula as to how often drops had to be used in each case in order that the tension might be maintained within safe levels. Of course, a patient who needed too frequent instillations was sent back to the service from which he was originally referred, for operative procedures.

A glandular extract and a new drug were used in some cases. These have been employed for too short a time and in too small a series of cases to warrant any definite conclusions.

As the purpose of the clinic was not only diagnosis and treatment but also research work along clinical lines, a problem from among the many confronting the clinic was allotted to each of the members of the staff.

Almost at the beginning of the work it was found that most of the patients did not understand the nature of their ailment and the importance of full cooperation. Naturally, limitation of time in the average clinic makes detailed instruction in this direction impossible. Most of the patients do not know how to instil drops, nor are they aware that sitting for a few hours in the dark (as in motion picture theaters), becoming excited, neglecting chronic colds in the head, drinking much strong coffee, and other practices may counteract the salutary effect of miotics. A leaflet with instructions printed in large type was given to

each patient, and arrangements were made for the numerous extra-medical problems to be handled by a well trained social worker. The National Society for the Prevention of Blindness cooperated in this matter. This society has agreed to supplement the salary of a social worker for the period of one year as a demonstration of what might be accomplished by social case work and follow up.

Almost from the outset the rapid accumulation of clinical data showed the necessity of systematization of records for the sake of ready reference and to make possible future statistical studies. After a number of revisions the final form adopted consisted of six record charts: Charts I and II are for the ocular and the medical history, respectively, and chart III, for the data obtained by examination of the eyes. Chart IV is for a systematic record of the subsequent course, which is checked on each revisit. On chart V is registered the social factors which may have a bearing on the disease, and chart VI contains a summary of the entire clinical picture and an outline of the projected course of treatment.

These charts have been kept as concise as was practicably possible and in actual use have been found to be the means of saving a great deal of time, once one has become familiar with them.¹ The two most useful (charts I and IV) are reproduced here, showing the history and the follow up in a typical case.

In chart I (fig. 1) the data in the first four columns in section E indicate that in 1928 the patient began to experience slight (1 +) fogging of vision and halos in the left eye, and in 1930 there were marked (4 +) loss of vision, ocular pain and headache with nausea and vomiting; that in November 1934 she noticed slight (1 +) fogging of vision, pain in the supra-orbital region and halos in the right eye, and that in July 1935 she became aware of diminished visual acuity and frequently stumbled over objects of furniture.

The next two columns (F and G) give more information about the aforementioned symptoms, correlated with the numbered lists of aggravating and relieving factors. It is seen that the patient's symptoms were aggravated at certain times of the day (column F, 1), when she was worried (column F, 4) and during colds in the head (column F, 6), but were relieved by rest (column G, 22) and hot applications (column G, 26).

The two columns to the left of column F are for details of the first and last acute attacks. In this case there was only one attack, in the left eye, in 1930, lasting seventy-two hours, brought on by "trouble

1. The space is too limited to allow the publication of the records and explanatory notes. We shall be glad to mail an exemplar of each to those desiring to obtain them.

at home" (column F, 5) and relieved only by enucleation (column G, 31). The attack consisted of 4 + loss of vision, 4 + ocular pain, 4 + headache and 2 + nausea and vomiting.

Section H indicates that the glaucoma was discovered in 1930 (the time of the patient's acute attack in another clinic and that she was given drops for the right eye and advised to have an operation on the left eye. It is also learned that she had been to an optometrist in 1928 and 1929, and a glance back at column E will recall that the fogging of vision and the halos in the left eye started in 1928. "(Over)" refers to a space on the reverse of the chart, left blank for the addition of

GLAUCOMA RECORD - EYE HISTORY															I		CLINIC NO 209412							
A. NAME		a R		AGE	51	SEX	F	M	S	D	SEP.	CLINIC	Knapp		SERIAL NO									
ADDRESS		642 a Ave. Bx.		OCCUP.		Chambermaid		RACE		Wh		SERVICE OF		Dr. Knapp		DATE OF HISTORY		Nov. 12, 1935						
B. DIAGNOSES															C. OPERATIONS AND DATES									
1. EYE { PRINCIPAL O.D. Chr. Simple Glaucoma O.S. Anophthalmos SECONDARY O.D. Hyp. Astig. Presby. O.S.															O.D. — O.S. Strabotomy (?) 1930 Enucleation 1930									
2. GENERAL Hypertthyroidism, Sec. Anemia																								
D. CHIEF COMPLAINT															H. GLAUCOMA DISCOVERED									
O.D. Vision poor, 4 no. Headaches and foggy vision for past year.															DATE 1930 BY whom other clinic ADVISED: drops O.D. operation O.S. DATE SEEN 1928, 1929 GLAUCOMA DISCOVERED BY whom optometr. DROPS STARTED (DATE) R: 1930 (over) L: — AT PRESENT USING MO DROPS FREQUENCY R: L: TOTAL # ACUTE ATTACKS R: 0 L: 1									
E. GLAUCOMA SYMPTOMS															F. SYMPTOMS BROUGHT ON BY					G. SYMPTOMS RELIEVED BY				
															1. TIME OF DAY					21. SLEEP				
															2. FATIGUE					22. REST				
															3. USE OF EYES					23. SEBATIVE				
															4. WORRY					24. EYE DROPS				
															5. TROUBLE AT HOME					25. COLD ATTK.				
															6. HEAD COLD					26. HOT APPLIC.				
															7. ILLNESS					27. CHANGE OF ENVIRONMENT				
															8. CONSTIP. OR DIGESTIVE UPSET					28. HOSPITALIZED				
															9. MENSES					29. OPERATION				
															10. WEATHER					30. SPONTANEOUS				
															11. NO KNOWN CAUSE					31. ————				
															12. ————					32. ————				
															13. ————					33. ————				
USE REVERSE, FOR ADDITIONAL DATA AND DETAILS															SIGNATURE.					B.E. M.D.				

Fig. 1.—Chart for the optical history.

notes, comments and sketches. Here it is stated: "Patient spontaneously discontinued drops and clinic visits in 1931 because she felt well"—an all too frequent occurrence in the absence of social service. On admission to the clinic of the Herman Knapp Memorial Eye Hospital the patient had been using no drops.

Having read all this, one can readily appreciate the value of being able to record so detailed a history on two thirds of an ordinary 8 by 5 inch (20.3 by 12.7 cm.) clinic card.

Chart IV (fig. 2) is almost self-explanatory. One line is used for each revisit and tells the facts noted as a routine each time. Any notes outside these may be added on the reverse side of the chart, left blank

for such purposes. Column 35 may be used for writing in an appropriate heading to keep a record of some special measure indicated and being tried in a case. Column 39 calls for a check or an initial each time that perimetric examination is carried out, making for greater regularity in field taking.

A good deal of effort was also spent in preparing a blank for recording the medical and psychologic abnormalities of the patient and, when possible, of the members of their families also. Dr. A. Baumann, the medical diagnostician of the glaucoma clinic, has worked out a record which we deem to be as complete as possible and helpful in cor-

NAME		GLAUCOMA — SUBSEQUENT RECORD IV										CLINIC NO. 209412	
a. R												SERIAL NO.	
30	31. VISION WITH BEST CORR.		32. MEDICATION		33. HOURS SINCE LAST MED.		34. TENSION mm.		35.	36.	37. REMARKS	38. CLINICIAN	39. PROPERTY
DATE	R	L	R MED. FREQ.	L MED. FREQ.	R	L	R	L		R			
11-19-35	20/40	—	#6 4 x d	—	4	—	28	—		#6 5 x d	No fogging or halos	E	E
11-26	20/40+2	—	#6 5 x d	—	4	—	23	—		"	Basal metab.: +15	E	
12-3	20/40+2	—	#6 5 x d	—	4	—	23			"	R: her new gl.	E	
12-10	20/40+2	—	"	—	4		23			"		E	
12-17	20/40-3		"		4		23			(buffered)	Redness & tearing 2d. Conjunctivitis;	E	
12-31	20/40+1		" (buffered)		3		21			"	better since using buffered #6	E	B
1936 1-14	20/40+5		"		3 1/2		21			"		E	
1-28	20/40+2		"		20!		36!			"	Ran out of drops	E	
2-11	20/40		"		3 1/2		21			"		E	B
2-25	20/40+2		"		4		23			"		E	
3-17	20/50-2		"		4		30			#6 q 2 h. Bromides	Trouble at home (see over)	E	
3-24	20/40-1		#6 q 2 h. Bromides		4		23			"		E	
4-7	20/40		"		4		23			"		E	B
4-28	20/40+1		"		3 1/2		20			#6 4 x d. Disc. Bromides	New living quarters	E	
5-19	20/40		#6 4 x d		4		20			"	TSetter	E.	

Fig. 2.—Chart for the subsequent history.

relating the ocular findings with the general physical status. A number of interesting and new observations were thus made and will be ready for publication when the number of patients is larger.

In the course of the work of the clinic the staff was confronted by a number of questions and difficulties which could not be answered or overcome without the cooperation of all the other ophthalmic institutions of New York.

The first question was: What are the distant results with the present day methods of examination and treatment of patients with glaucoma? Then, in what percentage of cases of glaucoma have ophthalmologists

succeeded in conserving useful vision during the past ten years? Does their work on glaucoma show better visual results during the past ten years than it did during the preceding decade?

Some who have been practicing ophthalmology for several decades doubt whether the results in glaucoma in the period from 1925 to 1935 are superior to those in 1915 to 1925. Whether or not this is so cannot be ascertained unless a thorough survey is made of the entire population with glaucoma in the dispensaries of New York. Such a survey is unquestionably of fundamental importance and must, of course, be made with all the care and accuracy necessary to secure reliable statistical data. Nowadays, the study of any problem—be it scientific, sociologic, commercial, etc.—is invariably begun by a careful appraisal of all the factors involved. A survey has the advantages of pointing out weak points or trends that lead nowhere or in wrong directions, faulty methods and other weaknesses.

The next question that arose was: Judging from our year's experience in the glaucoma clinic, do we feel that the patients are better handled, their diagnoses more completely worked out and the indications for various types of treatment more accurately established in a separate department than in the old-fashioned general clinic? The following facts are overwhelmingly in favor of special glaucoma clinics:

1. Among all the patients with ocular diseases, patients with glaucoma deserve to be placed in a special group. Their examinations are much more time consuming; each patient has to be carefully followed and studied. For this reason these patients cannot be treated in a routine fashion.

2. The psychologic approach to cases of this condition, in which mental upsets play an important rôle, must be performed with the same care and tenderness used in any surgical operation. In clinics for patients with thyroid disease a great deal of attention is given to this principle.

3. The social worker's function of disentangling complicated social and psychologic situations can be performed only in quiet and relaxing surroundings—not in the midst of the bustle of a general clinic.

4. Having to deal with a large number of patients with glaucoma, the ophthalmologists in the special clinic are given a splendid chance to acquire a vast amount of knowledge concerning the various phases of the problem of glaucoma, and those among them who have a bent toward research have a good opportunity to discover points previously unknown or to clear up questions previously misunderstood.

5. The patients quickly realize that special efforts are being made to help them, and they respond in a gratifying manner by cooperating to the full extent of their ability.

6. Year by year the carefully kept records grow in number and content and in the course of time furnish valuable material for study.

For these reasons, among others, we thought it advisable to call the attention of some of the ophthalmologists of New York to the advantages and possibilities of special clinics for patients with glaucoma. The response of our confrères has been prompt and favorable. Within a short time two colleagues have signified their intention to open special glaucoma clinics in other institutions, and there is no doubt in our minds that in due time others will join the ranks.

The idea of special clinics is not an entirely new one in medicine. There are already numerous clinics for persons with cardiovascular disease, diabetes, cancer, arthritis and other diseases. Many of those who are attending such clinics have formed local or national associations for study and exchange of ideas regarding the diseases of their special field.

Ultraspecialization in medicine is deprecated nowadays; the modern trend is toward correlation of all the findings furnished by a complete examination of the whole patient and, whenever desirable, of the whole family. By the same token patients with glaucoma should no longer be treated as though the only ailment was a pair of sick eyes for which but one of two things may be done: operation or the administration of miotics. Glaucoma clinics are meant to do for their patients what clinics for diabetes or clinics for cardiovascular disease are doing for theirs. They are meant to evaluate the entire physical, mental and inherited make-up of the patient, reconstruct a complete picture of the problem and try to do something for the whole patient instead of for his eyes alone.

Finally, not only the patient but the ophthalmologist can profit considerably from the special clinic for patients with glaucoma. The opportunity to see and follow such a group of patients will result in rounding out a knowledge of glaucoma which could hardly be considered complete even among the best ophthalmologists.

SUMMARY

Our one year's experience with the glaucoma clinic of the Herman Knapp Memorial Eye Hospital has taught us that such a clinic is fulfilling an urgent need, that it is in step with the spirit of modern clinical medicine, that it is bound to serve the best interest of the patients and of the community by preventing blindness whenever possible and that it offers opportunities for research unequaled by the average ophthalmic clinic.

It is our firm belief that progress is possible, if the problem of glaucoma is attacked by:

(a) A systematic and intensive medical as well as ophthalmologic study of each patient with glaucoma.

(b) Making a thorough survey of the glaucoma material in the ophthalmic institutions of the metropolitan area.

(c) Establishing a glaucoma clinic in each ophthalmic institution.

(d) Organizing an association of glaucoma clinics.

667 Madison Avenue.

MODIFIED SUBCONJUNCTIVAL EXTRACTION OF CATARACT

A PRELIMINARY REPORT

H. C. ERNSTING, M.D.

CLEVELAND

Subconjunctival extraction of the lens is not a recently developed procedure. It was recommended and performed early in the nineteenth century and has been more or less continuously practiced since that time.

Many modifications of the original technic have been developed, and only recently W. Moehle¹ described a method of subconjunctival extraction of the lens. It is evident, from the literature, that a progressive attempt has been made to modify and improve the technic of this type of operation.

Since its origin this method has been recommended for those cases in which loss of vitreous is threatened either during or after extraction of the lens.

The procedure which I am presenting is a modification of subconjunctival extraction which retains the qualities for which the extraction has been recommended and at the same time permits ease and simplicity of operation.

Before operation a complete history is taken, and the patient is given a physical examination. The laboratory examination varies in extent with the condition of the patient, but examination of smears from the eye, urinalysis, determination of the blood chemistry and a Wassermann test are carried out as a routine in each case. Each eye is irrigated with a 1:8,000 aqueous solution of merthiolate three times daily, and a 1 per cent solution of atropine is instilled into the cataractous eye the night preceding the operation and again on the morning of the operation. Anesthesia is produced by six instillations, given at intervals of three minutes, of a 4 per cent solution of either cocaine or metycaine. Sedatives are given preoperatively. A binocular dressing of mercury bichloride is applied before the patient is taken to the operating room.

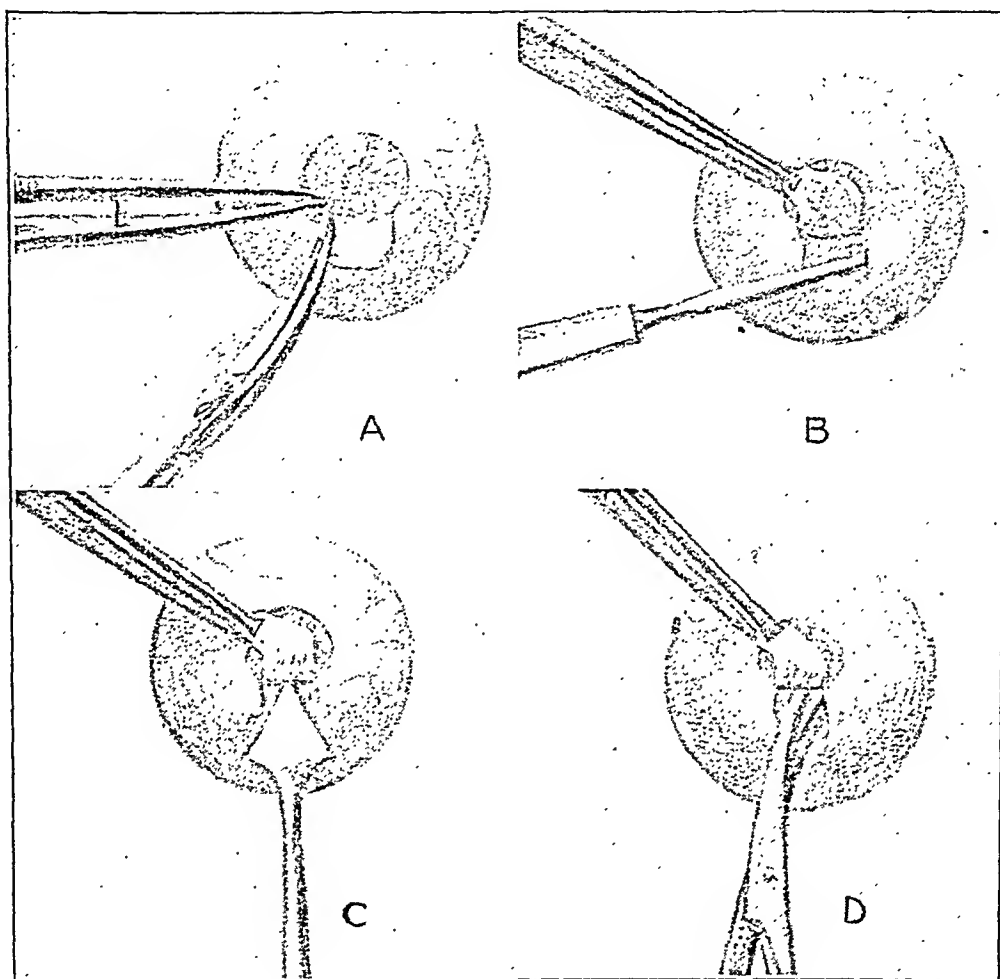
¹ From the service of Drs. W. E. Bruner, P. G. Moore, G. L. Miller and B. J. Wolpaw.

From the Department of Ophthalmology of the Western Reserve University School of Medicine and the Cleveland City Hospital.

1. Moehle, W.: Cataract Extraction Through a Vertical Conjunctival Slit, *Arch. Ophth.* **16**:659 (Oct.) 1936.

TECHNIC OF OPERATION

The field of operation is cleaned and draped in the usual manner, and akinesis is produced by means of the method of O'Brien² or that of Van Lint.³ The lid retractor is then inserted; the eye is irrigated with a 4 per cent solution of boric acid, and the patient is requested to look toward his feet so that adequate exposure of the superior part of the eye may be obtained.



A, a conjunctival flap approximately 6 by 4 mm. is made at 12 o'clock. This is dissected free from the underlying tissues up to the limbus. *B*, the conjunctiva on each side of the flap is undermined up to the limbus to 3 and 9 o'clock. *C*, a keratome is placed at the limbus, beneath the conjunctival flap, at 12 o'clock and is inserted into the anterior chamber in the usual manner. *D*, the primary incision is enlarged on each side to the extent of the undermined conjunctiva by blunt-pointed scissors, one blade of which is placed in the anterior chamber just anterior to the iris and the other blade of which is placed outside but beneath the conjunctiva which has previously been undermined.

2. O'Brien, C. S.: Local Anesthesia, *Arch. Ophth.* **12**:240 (Aug.) 1934.

3. Van Lint: *Ann. d'ocul.* **151**:420, 1914; *Arch. d'opht.* **43**:714, 1926.

A conjunctival flap approximately 6 by 4 mm. is then made at 12 o'clock. This is dissected free from the underlying tissues up to the limbus and is laid over the cornea to keep it out of the way.

The conjunctiva on each side of the flap is undermined up to the limbus to 3 and 9 o'clock by means of an iris repositor.

A keratome is then placed beneath the conjunctival flap at 12 o'clock and inserted into the anterior chamber in the usual manner. The primary incision is enlarged on each side to the extent of the undermined conjunctiva by blunt-pointed seissors, one blade of which is placed in the anterior chamber just anterior to the iris and the other blade of which is placed outside but beneath the conjunctiva which has previously been undermined.

A peripheral or basal iridectomy is then done, and the lens is extracted. The anterior chamber may be irrigated, if necessary.

The pillars of the iris are repositioned, if necessary, and the conjunctival flap is replaced and smoothed over with a spatula. No sutures are required.

White's ointment⁴ and a 1 per cent solution of atropine are instilled into the conjunctival sac. The lids are gently closed after it is ascertained that the flap is not displaced, and a binocular dressing is applied.

COMMENT

I have presented this modification of the subconjunctival type of extraction of the lens because it permits adequate working space and ease and simplicity of performance, while at the same time it retains all the qualities for which this type of extraction of cataract is recommended.

4. White's ointment is prepared as follows: Mercury bichloride, 1:3,000, sodium chloride, 5:3,000, and an ophthalmic ointment base, to saturation.

PROGNOSIS OF POSTOPERATIVE SYMPATHETIC OPHTHALMIA

A STATISTICAL STUDY

HAROLD H. JOY, M.D.

SYRACUSE, N. Y.

There are many factors which make a study of collected cases of sympathetic ophthalmia difficult and somewhat unsatisfactory. The question of diagnosis is disturbing, as much depends on interpretation not only of the clinical signs but of the histologic picture. Unfortunately, histologic examination is so often omitted that the number of cases that can be collected in which the diagnosis has been confirmed is materially reduced. Then there are the questions of the type of therapy used and the time it was started, and the condition of the sympathizing eye when treatment was instituted. Furthermore, each case is an entity in itself, and the disorder may be dependent on entirely different factors from those in a similar disorder produced under the same conditions. This variation in the different factors which enter into sympathetic inflammation is confusing and unless carefully considered may lead to incorrect conclusions. Nevertheless, such a study may be of some value either in confirming the conclusions of other investigators or in demonstrating the possibility that no conclusions are possible in such a complex condition. In any event, it brings to the consciousness of ophthalmologic surgeons a hazard that is always present.

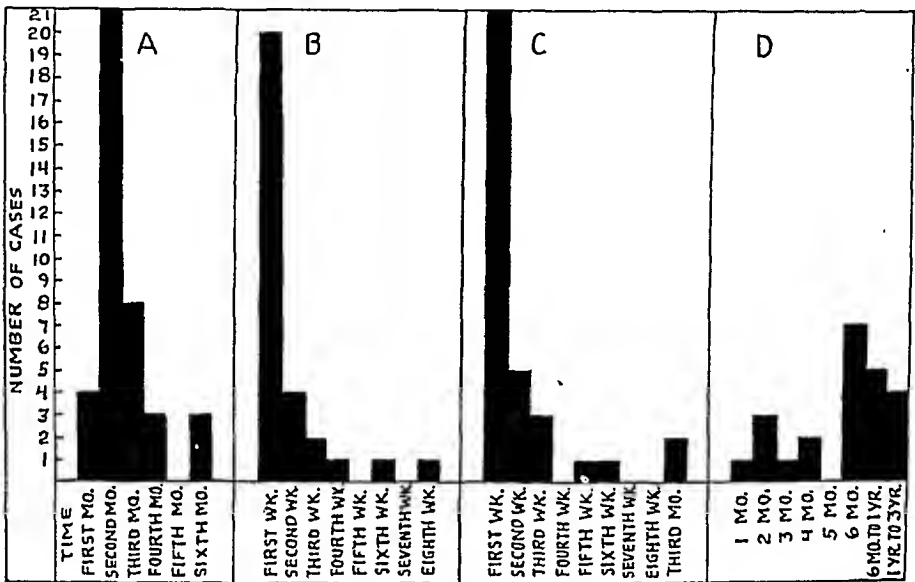
This study is based on one hundred and three cases of postoperative sympathetic ophthalmia collected by colleagues and from the literature. Chief consideration is given those cases in which the diagnosis was confirmed by microscopic examination. Of forty-four patients in whom an enucleated eye or a specimen of iris disclosed the characteristic histologic picture, one never showed sympathetic inflammation. Of the cases in which the diagnosis was proved in which the fellow eye later became involved, in twenty-six the disorder was attributed to operations for cataract, in sixteen it followed operation on the iris and in one it resulted from excision of the anterior segment of the globe.

Some other extenuating exciting factor was reported for twenty-five of the patients, in five of whom more than one such complication occurred. A definite preoperative pathologic process was observed in fifteen patients; an accident occurred during the operation or shortly

afterward in thirteen, and a second operation had been performed on ten. This phase has been discussed in another paper.¹

The interval between the operation and the onset of sympathetic inflammation is shown in section *A* of the chart. It ranged from a minimum of two weeks in two cases to a maximum of six months, with by far the greatest incidence (84.6 per cent) within the first three months, particularly during the second month (53.9 per cent).

The exciting eye was removed in thirty-nine patients, for six of whom the time of enucleation is not recorded. In three instances sympathetic ophthalmia developed in spite of prophylactic enucleation, while in the remaining thirty patients the exciting eye was removed as a



Diagrams showing relationships in time in the series studied. *A* shows the interval between operation and the onset of sympathetic ophthalmia; *B*, the interval between the onset and enucleation; *C*, the interval between the onset and the beginning of treatment, and *D*, the duration of the condition.

therapeutic measure within three months after the onset of sympathetic inflammation, and in twenty (66.7 per cent) it was removed within the first week after the onset (section *B* of the chart). In five patients the exciting eye was not enucleated, but in three of these the inflammation in the sympathizing eye was so severe that it had to be removed.

Systemic therapy and treatment of the sympathizing eye were instituted within three months after the first signs of inflammation for all the thirty-five patients whose records are complete, and for twenty-six (74.3 per cent) they were begun within the first two weeks after the onset (section *C* of the chart).

1. Joy, H. H.: New York State J. Med. 36:1498 (Oct. 15) 1936.

The condition in the sympathizing eye appeared mainly as iridocyclitis, except in three cases, in which it involved predominantly the posterior part of the uvea. The course of the disease was designated in twenty-six instances; it was reported as severe in fourteen patients, moderate in eight and mild in four. The duration ranged from one month to three years (section *D* of the chart), and, as would be expected, it was six months or more in the majority of cases (69.5 per cent).

Table 1 shows the relationship between the time at which therapy was begun and the outcome. In the few instances in which there was variation between the time of enucleation and that of starting other treatment the time of the first intervention is used to indicate the time of the beginning of therapy. All other factors being disregarded, this table shows a much greater proportion of good results in those patients

TABLE 1.—*Relationship Between Outcome and Time After Onset That Therapy Was Instituted*

Time After Onset That Therapy Was Instituted	Number of Cases	Outcome	
		Successful	Unsuccessful
Within 2 days.....	4	3	1
From 3 days to 1 week.....	17	12	5
Second week	5	4	1
First month	4	1	3
Second month	2	2	0
Third month	2	0	2
Definite interval not stated; within 3 months.....	2	1	1
Questionable	7	?	7
Total number of cases.....	43	23	20

who received prompt treatment. Thus, useful vision resulted in 73 per cent of the patients in whom treatment was started within two weeks after the onset, compared with 37.5 per cent of those in whom it was delayed one month or more. Altogether, of thirty-eight patients whose final vision was recorded, the outcome was satisfactory in twenty-one (55.2 per cent); in thirteen instances vision was 20/40 or better (table 2). Of the seventeen patients in whom treatment gave unsuccessful results, one retained vision of 1/25, while in the others it was reduced to light perception or total blindness.

Various authors² emphatically have maintained that sympathetic ophthalmia following extraction of cataract is especially severe and the outcome unfavorable. This was found to be true in the five cases in which the diagnosis was confirmed, reported by me,³ and in one of

2. (a) Hambresin, M. L.: Bull. Soc. belge d'opht., no. 57, Nov. 25, 1928, p. 107. (b) Davids, H.: Arch. f. Augenh. **105**:13 (Oct.) 1931. (c) Jaeger, E.: Klin. Monatsbl. f. Augenh. **73**:714, 1924.

3. Joy, H. H.: A Survey of Cases of Sympathetic Ophthalmia Occurring in New York State, Arch. Ophth. **14**:733 (Nov.) 1935.

Woods' ⁴ two cases. Also, in E. Fuchs' ⁵ cases the condition was severe in four and moderately severe in one. On the other hand, Verhoeff ⁶ stated the belief that there are too many variable factors involved in this disease to enable one to arrive at a decision. Of the twenty-six cases in this series in which sympathetic ophthalmia resulted from operations for cataract, in nineteen the condition followed combined extraction of cataract of the senile type (table 3). All the patients but two were over 50 years of age. The incubation period ranged from four weeks to six months, and the duration from three to eighteen months. The condition in the sympathizing eye appeared mainly as iridocyclitis except in one case, in which it involved predominantly the posterior part of the uvea. In six patients in whom the exciting eye was enucleated and treatment was instituted within twelve days after the onset of sympathetic inflammation the outcome was uniformly good, all the patients attaining useful vision, while of three patients in whom the intervention was delayed from nineteen days to two months after the onset,

TABLE 2.—*Final Vision*

Vision	Number of Cases	Percentage
20/40 - 20/15.....	13	34.2 }
20/200 - 20/50.....	8	21.0 } 55.2
Perception of light - perception of fingers.....	5	13.2 }
No perception of light.....	12	31.6 } 44.8

only one showed successful results. The exciting eye was removed in two patients two weeks and one month, respectively, before any signs of inflammation were observed in the fellow eye, but blindness resulted in both after a moderately severe course. The exciting eye was not removed in four patients (table 4). In one, in whom the diagnosis was made from a specimen of iris, both eyes escaped with useful vision, despite a delay of forty-two days in starting treatment. In the other three patients the sympathizing eye was so severely involved that it had to be removed. This also occurred in three of the cases in which the diagnosis was unconfirmed. Jaeger,^{2c} in reporting thirteen of these rare cases, gathered from the literature and from the Tübingen clinic, found that in all except two the condition was due to extraction of cataract. In all three of the cases of this nature reported in the present series the condition followed multiple operations with complications. The incubation period was comparatively long, and there was a delay in starting treatment in at least two of them. In spite of the malignant

4. Woods, A. C.: *Am. J. Ophth.* **19**:9 (Jan.) 1936.

5. Fuchs, E.: *Arch. f. Ophth.* **61**:365, 1905.

6. Verhoeff, F. H.: Personal communication to the author.

course in the sympathizing eye, useful vision was attained in the exciting eye in each instance. This was also true in two of the cases in which the condition was unproved.

Such instances tend to substantiate the statements of those who maintain that the inflammation is disproportionately more severe in the sympathizing eye after extraction of cataract and are therefore reluctant to enucleate the exciting eye.^{2a} The data for twelve cases in which the condition was clinically diagnosed (table 5) in which neither eye was enucleated are not convincing. While better vision resulted in the exciting eye in five patients, there was a marked difference in only one. In two patients vision in the sympathizing eye was slightly better than that in the exciting eye, and in three patients there was no difference. All in all, the outcome was generally poor in both eyes, only three patients attaining useful vision. While there may be too many extenuating factors to warrant the conclusion that these data indicate the value of enucleation, they do not indicate any disproportion in the severity of the condition in the two eyes, nor do they indicate the value of retention of the exciting eye.

Of seventeen cases of sympathetic ophthalmia following combined extraction of cataract in which the outcome is recorded, there was complete loss of vision in the sympathizing eye in nine (53 per cent) (table 6). Useful vision was attained in eight cases (47 per cent), and in five of these it was 20/40 or better. While this represents the final vision in the sympathizing eye, it does not tell the complete story, for in the three patients in whom the sympathizing eye was enucleated useful vision resulted in the exciting eye. Therefore, eleven patients (64.7 per cent) actually attained useful vision. When thirty-one cases in which the condition was clinically diagnosed are included the data do not show such a favorable result. Considering the whole group of forty-seven cases, useful vision in the sympathizing eye resulted in nineteen (34 per cent). It is probable that both the figures for the cases in which the diagnosis was confirmed and those for the total group lead one to overestimate the damage caused by the inflammation, for in many instances the final vision was undoubtedly influenced by the presence of a cataract which existed before the uveitis appeared.

While no conclusions can be drawn from the data for this group of cases, the results of the study at least indicate that if proper treatment is promptly instituted sympathetic ophthalmia following combined extraction of cataract is not necessarily unusually violent and that the prognosis may not be as hopeless as many authors have indicated.

Sympathetic ophthalmia followed operations for congenital cataract in three patients (table 7). Two were children, and one was 29 years of age. The operations, consisting of a discission in two instances and a linear extraction in one, were all followed by a severe reaction. A

TABLE 3.—Data for Cases of Sympathetic Ophthalmia Following Combined Extraction of Cataract

Case	Reference	Age of Patient, Years	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Treatment	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, at Onset	Course	Duration	Outcome and Comment
346	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	54	Hypophymion 2 days after operation	10 wk.	1 wk.	1 wk.	Vision 6/12; incipient enteric; plastic iridocyclitis	Moderate	Several months	Vision 6/12; favorably influenced by operation
348	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	?	?	?	?	?	?	?	?	Blind
343	Post, L. T.: Personal communication to the author in regard to the cases compiled by D. H. Trowbridge	?	?	?	?	?	?	?	?	?
353	Moran, V.: Ann. d'oeul. 154: 705 (Dec.) 1917	73	Followed by Iridocyclitis	59 days	1 day	1 day	Iridocyclitis	?	?	Vision 5/10
261	Theobald, G. D.: Am. J. Ophth. 13: 597 (July) 1930	52	Followed by infection	4 wk.	A few days	6 days	Vision 20/200	Moderate	?	Vision 20/200
262	Theobald, G. D.: Am. J. Ophth. 13: 597 (July) 1930	56	Extraction of cataract attempted	8 wk.	Within 19 days	19 days	Very severe	Severe	?	?
307	Verhoeff and Irvine ¹⁰	41	?	10 wk.	12 days	1 wk.	Slight congestion; keratitis punctata; Koeppe nodules	?	1 yr.	Vision 20/30; 1½ yr. after subsidence; extraction of cataract in sympathizing eye 1½ yr. after onset
309	Verhoeff and Irvine ¹⁰	53	?	8 wk.	8 days	1 wk.	Slight congestion; a few synechiae pupillary exudate; vision 20/50	?	6 mo.	Vision 20/100 8 yr. after subsidence (poor vision due to senile cataract)

310	Verhoeff and Irvine ¹⁰	70	?	8 wk.	30 days	8 wk.	Vision 1/200; slight engorgement; keratitis punctata cataract	?	6 mo.	Vision 20/30 1 yr. after subsidence; extraction of cataract in sympathizing eye 6 mo. after onset
305	Woods, A. C.: Personal communication to the author	70.	?	2 mo.	3 wk.	At onset	Vision 20/100; involvement of posterior part of uvea	?	?	Vision 20/30
57	Joy ³	67	Complicated cataract; secondary glaucoma	30 days	Within 3 wk.	19 days	Loss in visual field; iridocyclitis	Moderate	3 mo.	No perception of light
8	Joy ³	65	?	From 6 to 10 wk.	?	?	?	Severe	?	No perception of light
101	Joy ³	60	High myopia	3 mo.	?	?	Plastic uveitis with exudate; secondary glaucoma	Severe	?	No perception of light
67	Joy ³	68	Uveitis	6 mo.	Prompt	1 mo. before	Plastic uveitis	Moderate	18 mo.	No perception of light
238	Wood, D. J.: Brit. J. Ophth. 11: 217 (May) 1927	53	Abrasion of cornea several days after operation; dissection 7 wk. after extraction of cataract	74 days	A few days	Enucleation of sympathizing eye	Iridocyclitis; keratitis punctata	Severe	?	Sympathizing eye enucleated; vision of exciting eye 6/6, 1½ yr. later
222	Schreffer, L.: Arch. f. Ophth. 129: 127 (Sept.) 1932	66	Complicated cataract; dissection 3½ mo. later; secondary glaucoma	6 mo.	Within 2 mo.?	Enucleation of sympathizing eye	Iridocyclitis; secondary glaucoma	Severe	?	Sympathizing eye enucleated; vision of exciting eye 0.5, 2 yr. later
207	Jaeger, E.: Klin. Monatsbl. f. Augenh. 78: 613, 1927	42	Iris in scar; dissection 2 mo. after extraction	3½ mo.	2½ mo.	Enucleation of sympathizing eye	Faulty projection; iritis; keratitis punctata	Severe	1 yr.?	Sympathizing eye enucleated; vision of exciting eye 5/8, 1 yr. later
318	Verhoeff and Irvine ¹⁰	68	?	16 wk.	42 days	No enucleation	Immature cataract; keratitis punctata; total synechia	?	6 mo.	Vision of sympathizing eye 20/200, vision of exciting eye 20/50, 1½ yr. after subsidence

subsequent linear extraction was necessary in two patients, and in one of these it resulted in prolapse of the iris and loss of vitreous. The incubation period ranged from seven weeks to six months. The exciting eye was removed and treatment begun promptly in all three cases; in

TABLE 4.—*Data for Cases in Which the Diagnosis Was Confirmed in Which the Exciting Eye Was Not Enucleated*

Case	Complications	Interval Between Operation and Onset	Interval Between Onset and Treatment	Course	Interval Between Onset and Enucleation of Sympathizing Eye	Outcome in Exciting Eye
318	?	16 wk.	42 days	?	Not enucleated	Vision of exciting eye 20/50, vision of sympathizing eye 20/200, 1½ yr. after subsidence
238	Yes	79 days	A few days	Severe	6½ mo.	Vision 6/6, 11¼ mo. after enucleation
232	Yes	6 mo.	Within 3 mo.	Severe	3 mo.	Vision 0.5, 2 yr. after enucleation
207	Yes	3½ mo.	2½ mo.	Severe	1¼ yr.	Vision 5/8

TABLE 5.—*Data for Cases of Clinically Diagnosed Sympathetic Ophthalmia Following Extraction of Cataract in Which Neither Eye Was Enucleated*

Case	Exciting Eye	Sympathizing Eye
132.....	Perception of light	No perception of light
131.....	Vision 20/200	No perception of light
111½.....	Perception of light	No perception of light
105.....	Perception of fingers	No perception of light
259.....	Vision 1/60	Perception of light
300.....	Perception of movements of hands at 4 in.	Perception of movements of hands at 14 in.
338.....	Perception of light	Vision 4/200
389.....	Perception of light	Perception of light
42.....	Vision 6/12	Vision 6/12
33.....	No perception of light	No perception of light
272.....	?	No perception of light
335.....	?	Vision 20/20

TABLE 6.—*Final Vision in Cases of Sympathetic Ophthalmia Following Combined Extractions*

	Number of Cases	Percentage
20 /40 - 20/15.....	5	29.4
20/200 - 20/50.....	3	17.6
Perception of light - perception of movements of hands...	1	6.0
No perception of light.....	8	47.0

two of them treatment was started within one day after the onset of sympathetic inflammation. The duration was short, varying from one month to four months. Two of the patients obtained useful vision, and one became blind. In a case of clinically diagnosed sympathetic ophthalmia in which the exciting eye was not removed the outcome

TABLE 7.—Data for Cases of Sympathetic Ophthalmia Following Operations for Congenital Cataract

Case	Reference	Age of Patient, Years	Type of Operation	Complications	Interval Between Onset of Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision When Patient Was Seen	Course	Duration	Outcome and Comment
243	Case compiled by the author in the Wills Eye Hospital	29	Linear extracapsular	Large amount of lenticular cortex; linear extraction repeated 3 wk. later	7 wk.	1 day	"Mutton-fat" deposits of keratitis punctata; iris congested; vision 6/25	Mild	1 mo.	Vision 6/25; gradual improvement after enucleation
116	Joy ³	3½	Dissection	Hyphemia; exudate in anterior chamber; tension +; no perception of light	6 mo.	Less than 3 mo.	?	Moderate	2 mo.	Vision 20/40
314	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	8	Dissection	Lens in anterior chamber; glaucoma; lens extracted 1 wk. later, with loss of vitreous and prolapse of the iris	9 wk.	Same day	Keratitis punctata; pupil contracted	Severe	4 mo.	Blind; enucleation did not influence progress

TABLE 8.—Data for Cases of Sympathetic Ophthalmia Following Extraction of Traumatic Cataract

Case	Reference	Age of Patient, Years	Type of Injury	Interval Between Injury and Operation	Type of Operation	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Onset of Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, and Vision, When Patient Was First Seen	Course	Duration	Outcome and Comment
240	van Schievensteen, A., Jr.: Ann. d'ocul. 155: 392 (Aug.) 1918	31	Perforating wound; foreign body in iris	3 mo.	Extraction of cataract	Patient squeezed at operation, causing prolapse of iris and hyphema	49 days	3 days	Vision 0.4; ciliary congestion; tension —	?	6 mo.	Cured
244	Oguchi: Acta soc. ophth. jap. 38: 98 (June) 1934	39	Blow from piece of wood	?	Extraction of cataract	?	2 mo.	?	Iritis and neuroretinitis	?	?	Did not improve
13	Joy ³	27	Perforation; foreign body in lens	6 yr.	Combined extraction in capsule	Secondary glaucoma and low grade uveitis before operation	7 wk.	A few days	Vision 20/200; severe iridocyclitis	Severe	5 wk.	Vision 20/15
9 M.	Morax, V.: Ann. d'ocul. 134: 705 (Dec.) 1917	26	Injury to iris	2 wk.	Extraction of cataract	Prolapse of iris before operation	58 days.	Prompt	Severe iridocyclitis	Severe	?	Vision 1/25

was poor, with vision of perception of fingers at 2 feet (60 cm.) in the sympathizing eye and loss of perception of light in the eye on which the operation had been performed.

Perforation of the globe at the time of injury had occurred in three of the four cases of sympathetic inflammation following operations for traumatic cataract (table 8). This had resulted in prolapse of the iris in one instance, while in two an intra-ocular foreign body was present at the time of the extraction. The patients were all men from 26 to 39 years of age. The interval between the injury and the operation ranged from two weeks to six years, and with one exception the eye was quiet at the time of operation. In the latter patient low grade uveitis and secondary glaucoma had developed as the result of a perforating wound six years before. Another patient misbehaved during the operation, causing prolapse of iris and a large hyphemia. The incubation period ranged from seven weeks to two months. In at least three of the cases treatment was instituted promptly and the exciting eye enucleated within a few days after the onset. Two of the patients made a satisfactory recovery, and two did badly, while in five cases in which sympathetic ophthalmia was clinically diagnosed three patients attained useful vision.

Of the fifteen cases of sympathetic ophthalmia following operations on the iris, in all but one the condition was attributed to some form of operation for glaucoma (table 9). The exception was a case in which a repair operation was performed for iridodialysis five weeks after a severe contusion. Among the remaining cases, in six the condition occurred after iridectomy, in five it followed operation for iris inclusion, and in three it was due to trephining, while in one case the type of operation was not recorded (table 10). With four exceptions the patients were 40 years of age or older. The incubation period ranged from thirty-three days to four months. Treatment was instituted within two weeks after the onset in twelve cases and within three months after the onset in all. The duration extended from two months to three years. Jaeger^{2c} stated that sympathetic ophthalmia does not often follow iridectomy and when it does it is often of a mild type. This was not true in the six cases in this series, for although the exciting eye was removed and treatment begun within two weeks after the onset of the first symptoms, the course was either moderate or severe. Although four patients ultimately attained useful vision, the uveitis was still active in one when the case was reported. In two cases in which the condition was clinically diagnosed the outcome was satisfactory. Peters,⁷ in reviewing the reported cases in which sympathetic ophthal-

7. Peters, A.: *Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten*, Ztschr. f. Augenh., supp. 20, 1936.

TABLE 9.—Data for Cases of Sympathetic Ophthalmia Following Operation on the Iris

Case	Reference	Age of Patient, Years	Type of Operation	Complications	Interval Between Operation and Onset of Sympathetic Ophthalmia	Interval Between Sympathetic Ophthalmia and Enucleation	Character of Sympathetic Ophthalmia, at Onset	Course	Duration	Outcome and Comment
					7 wk.	Prompt				
88	Joy ³	25	Iridodialysis as a repair operation	Operation done 5 wk. after severe contusion	7 wk.	Prompt	Prompt	Severe	6 mo.	Vision 20/15—
Operations for Glaucoma										
341	Higbee, E. H.: <i>Am. J. Ophth.</i> 9: 584, 1926	42	Iridectomy for secondary glaucoma	Iritis before operation	No sympathetic ophthalmia	No sympathetic ophthalmia	No sympathetic ophthalmia	Vision 20/20; no sympathetic ophthalmia at any time
220	Groenouw, A.: <i>Klin. Monatsbl. f. Augenh.</i> 76: 604, 1926	?	Iridectomy	Prolapse of Iris	5 wk.	Within 2 wk.	Disseminated choroiditis; falling vision	Moderate	4½ mo.	Vision, part of 5/15
247	Gifford, S. R., and Lucie, L. H.: <i>Tr. Sect. Ophth., A. M. A.</i> , 1926, p. 20	70	Iridectomy	Melanosarcoma of choroid	5 wk.	5 days	Perception of fingers at 5 in.; rapidly progressive irvetitis; exudate in pupil	Severe	?	Vision 6/30
327	Verhoeff and Irvine ¹⁰	61	Iridectomy	?	7 wk.	5 days	Vision 20/50; moderate congestion; many deposits of keratitis punctata; posterior synechia	?	6 mo.	Vision, perception of light; inflammation inactive for 6 mo.
342	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	60	Iridectomy	Iritis on sixteenth day; hypopyon on twenty-fourth day	33 days	8 days	Vision 20/30—perception of fingers at 10 ft. 3 days later; tension +; deposits on anterior capsule	Moderate	1 yr. +	Vision 20/20; inflammation still active; progress favorably influenced by enucleation
345	Post, L. T.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	67	Iridectomy	None; low grade iridocyclitis 1 mo. later	7 wk.	2 wk.	Almost total posterior synechia; secondary glaucoma; blurred vision	Severe	8 mo.	Blind; progress not influenced by enucleation; patient died

		69	Iridectomy	Gaping wound; ecstasis of iris; vitreous pre- senting	11 wk.	1 wk.	1 wk.	1 wk.	Vision 20/15, 1 yr. after onset; involvement of the anterior and pos- terior part of the uvea	?	3 yr. ?	Subsided
304	Woods, A. C.: Personal communication to the author											
231	Schomberger, H.: Arch. f. Ophth. 125:28, 1930	50	Trephna- tion	Sclerotomy neces- sary 8 days later; early iridocyclitis	1 mo.	Prompt	Prompt	Prompt	?	?	?	Total loss of vision
263	Theobald, G. D.: Am. J. Ophth. 13:597 (July) 1930	62	Trephna- tion	Iridectomy	15 wk.	4 days	4 days	4 days	?	Moder- ate	?	Vision 20/20
9	Joy ²	23	Trephna- tion	Trephination repeated	4 mo.	Less than 3 mo.	Less than 3 mo.	Less than 3 mo.	Vision 20/40	Severe	From 3 to 4 yr.	Vision 20/70
249	Gifford, S. R., and Lucie, L. H.: Arch. Ophth. 1:468 (April) 1929	A	Iridencleisis	Large subconjunc- tival prolapse of iris and of ciliary body; part of which was excised later	?	A few days	A few days	A few days	Vision blurred; cells; disk blurred	Moder- ate	?	Vision 20/20—; eye cleared rapidly and remained quiet
317	Post, L. P.: Personal communication to the author in regard to cases compiled by D. H. Trowbridge	28	Iridencleisis on right and left eye for secondary glaucoma	None	9 wk.	At onset	At onset	6 wk.	Keratitis punctata; cells in anterior cham- ber; fundus normal; blurred vision	Moder- ate	2 mo.	Vision 6/4—2; progress favor- ably influenced by enucleation
314	Verhoeff and Irvine ¹⁰	66	Iridotaxis	?	5 wk.	2 days	2 days	1 wk. before onset	Vision 20/50; marked congestion; heavy deposits of keratitis punctata; synechiae; vision decreased to 3/200	?	1 yr.	Vision 20/70— (poor vision due to senile cataract); inflammation; inactive 1 yr.
321	Verhoeff and Irvine ¹⁰	44	Iridotaxis	?	8 wk.	14 days	14 days	Enuclea- tion at onset	Vision 20/40; moderate congestion; a few deposits of keratitis punctata	?	3 yr.	Vision, perception of light; sec- ondary glaucoma; inflammation in- active 1 yr.
330	Verhoeff and Irvine ¹⁰	40	Iridotaxis	?	5 wk.	63 days	63 days	No enu- cleation	Vision 1/200; marked congestion; pupil- lary membrane	3 yr.	Vision in synop- tizing eye, percep- tion of light; no perception of light in exother eye (primary glau- coma); inflamma- tion apparently still active
139	Joy ²	62	Operation for secondary glaucoma	?	?	Late ?	Late ?	?	?	Severe	9 mo.	No perception of light

mia followed trephining operations, mentioned only nine and questioned the diagnosis in several of these. In all three of the cases in this series the condition followed a second operation. Useful vision resulted in two, as well as in two of the three cases in which the condition was clinically diagnosed. Operations for iris inclusion would seem to offer fertile ground for the development of sympathetic ophthalmia, but there are surprisingly few cases reported in the literature in which this condition followed such an operation, and in none of the one hundred and fifty-one cases of sympathetic ophthalmia in the series in New York State which I³ reported was it the exciting cause. Of the five cases reported here, in three the end-result of the operation was

TABLE 10.—*Data for Cases of Sympathetic Ophthalmia Following Operations for Glaucoma*

Type of Operation	Number of Cases	Outcome	
		Good	Bad
Iridectomy.....	6	4	2
Trephination.....	3	2	1
Operation for iris inclusion.....	5	3	2
Operation for glaucoma.....	1	0	1

TABLE 11.—*Final Vision in Cases of Sympathetic Ophthalmia Following Operations for Glaucoma*

Vision	Number of Cases	Percentage
20/40 - 2/15.....	5	33.3
20/200 - 20/50.....	4	26.7
Perception of light.....	3	20.0
No perception of light.....	3	20.0

successful, while in two vision was reduced to perception of light. In a case in which the condition was clinically diagnosed which followed bilateral iridotaxis, final vision in one eye was 20/20 and in the other was reduced to perception of shadows.

Obviously no conclusions can be drawn regarding the relationship of the type of operation and the ultimate outcome. However, in this series sympathetic inflammation due to iridectomy for glaucoma was not mild or apparently infrequent.

The outcome was generally more favorable when treatment was started promptly. Among twelve patients for whom treatment was instituted within two weeks after the onset of sympathetic inflammation, useful vision resulted in eight, and four became blind, while in three patients in whose cases there was a delay of six weeks or more, one attained useful vision, and two became blind. Of the fifteen proved

cases in which the condition followed operations for glaucoma (table 11) the outcome in nine (60 per cent) was satisfactory, while in six (40 per cent) it was poor. The final vision in several instances was unfavorably influenced by the presence of primary glaucoma or senile cataract.

SECONDARY OPERATIONS

Because of the great frequency of cataract and glaucoma in both eyes, irrespective of the uveitis, subsequent operations to improve vision would seem to be more often necessary in sympathetic inflammation due to operation than in that due to other causes. For the same reason the prognosis would appear to be less favorable. While the exciting eye tolerates operations well, the sympathizing eye is notoriously sensitive, even after a long period of quiescence. Woods⁸ found that pigment therapy immediately preceding the operation is of real value in diminishing the reaction and in promoting the healing. Verhoeff⁹ has shown that if the patient is kept under the influence of diphtheria antitoxin the eye can sometimes be safely operated on even before the subsidence of inflammation. However, he and Irvine¹⁰ warned that if the patient cannot be made sensitive to the antitoxin and if the treatment is not obviously beneficial, surgical intervention should be avoided.

Table 12 shows the data for those cases of postoperative sympathetic ophthalmia in which either the exciting eye or the sympathizing eye was operated on during or subsequent to the inflammation. In nine of the patients the exciting cause was extraction of cataract, and in two the condition was attributed to iridectomy. The sympathizing eye had already been removed in all four patients whose exciting eyes were operated on. The surgical intervention, consisting of discission in three instances and of iridotomy in one, was well tolerated in all the cases and resulted in improved vision. The surgical intervention on the sympathizing eye was naturally more complicated, and in three patients more than one operation was performed, despite which there was an actually unfavorable outcome in only one case. The three cases in which the diagnosis was confirmed in which the sympathizing eye was operated on were Verhoeff's, whose unusually good results with diphtheria antitoxin were reported in 1935.¹⁰

8. Woods, A. C., quoted by de Schweinitz, G. E.: *Tr. Ophth. Soc. U. Kingdom* **46**:257, 1926.

9. Verhoeff, F. H.: *Arch. Ophth.* **56**:28 (Jan.) 1927.

10. Verhoeff, F. H., and Irvine, S. R.: *New York State J. Med.* **36**:63 (Jan. 15) 1936.

TABLE 12.—Data for Cases of Postoperative Sympathetic Ophthalmia in Which Either the Exciting Eye or the Sympathizing Eye Was Operated On

Case	Reference	Age of Patient, Years	Exciting Cause of Sympathetic Ophthalmia	Type of Secondary Operation		Time of Second Operation	Effect of Operation	Outcome
				Exciting Eye	Sympathizing Eye			
Cases in Which the Diagnosis Was Confirmed								
232	Schreibler, L.: Arch. f. Ophth. 129: 127 (Sept.) 1932	66	Extraction of complicated cataract	Dissection	1 yr. after subsidence	Favorable	Vision 0.5
307	Verhoeff and Irvine ¹⁰	41	Extraction of cataract	Extraction of cataract	6 mo. after subsidence	Favorable	Vision 20/30
310	Verhoeff and Irvine ¹⁰	70	Extraction of cataract	Extraction of cataract	At subsidence	Favorable	Vision 20/30
327	Verhoeff and Irvine ¹⁰	61	Iridectomy	Operation	?	Unsuccessful	Vision, perception of light
Cases in Which the Condition Was Clinically Diagnosed								
258	Extraction of cataract	Dissection	?	Favorable	Useful vision
275	Hambreslin ^{2a}	?	Extraction of cataract	Dissection	Shortly after subsidence	Favorable	Useful vision
105	Joy ³	Adult	Extraction of cataract	Iridotomy	Shortly after subsidence	Favorable	Patient able to get about
232	Veidovsky, V.: Oftal. Sborn. 7: 109, 1932	?	Extraction of cataract	Preliminary iridectomy; extraction of cataract; capsulotomy	?	Comparatively favorable	Vision, perception of fingers at 40 cm.
274	True, H., and Dejean, C.: Bull. Soc. franç. d'opt. 38: 646, 1925	72	Extraction of cataract	Preliminary iridectomy; extraction of cataract	About 2 mo. after subsidence	Comparatively favorable	Vision, counting fingers
260	Cases compiled by the author in the Willis Eye Hospital	8	Linear extraction combined with iridectomy	Capsulotomy; paracentesis; iridectomy	During course of uveitis	Comparatively favorable	Vision, perception of fingers at 1.5 meters
333	Verhoeff and Irvine ¹⁰	45	Iridectomy	Iridectomy	?	Favorable	Vision 20/20

SUMMARY

Sympathetic ophthalmia is too complicated a disease to permit one to draw conclusions from such a small number of cases. However, study of this series brings out several points worthy of notice.

1. The final visual results indicate that the prognosis of postoperative sympathetic ophthalmia is not necessarily as unfavorable as many authors have stated, provided proper treatment is instituted with promptness.

2. The final outcome in the cases in which the condition followed combined extraction of senile cataract was less favorable than in those in which it followed other intra-ocular operations.

3. The inflammation in the sympathizing eye was disproportionately more severe than that in the exciting eye in three cases of sympathetic ophthalmia due to extraction of cataract in which the diagnosis was confirmed and three cases of the same kind in which the condition was clinically diagnosed. However, this was not apparent in twelve cases in which the condition was clinically diagnosed in which neither eye was enucleated.

4. There is no indication that sympathetic ophthalmia due to iridectomy for glaucoma is particularly rare or that its course is mild.

5. The results in the few instances in which secondary operations were performed indicate that the exciting eye tolerates surgical intervention well and that the sympathizing eye can often be safely operated on if it is properly prepared for the intervention.

504 State Tower Building.

Alan C. Woods presented the facilities of the library of the Wilmer Ophthalmological Institute for use in making this study.

OPTIC ENCEPHALOMYELITIS

REPORT OF A CASE

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Optic encephalomyelitis is comparatively rare. The object of this paper is to report another case of this disease that presented features of special interest.

REPORT OF CASE

History.—Mrs. R. O., aged 34 years, came to me on Nov. 20, 1932, complaining of a feeling of pressure and of loss of vision in the right eye, of about two weeks' duration.

There was no history of diseases of childhood, but the patient had pneumonia five years previously and had had occasional sore throat. She suffered from dyspnea on exertion; swelling of the ankles, especially when on her feet for any length of time, and occasional dizzy spells. Her gallbladder had been removed nine years before, but she still complained of gas and of discomfort in the abdomen and epigastrium after meals. There was no history of jaundice. The family history was not important.

Physical Examination.—The patient was sent to the Royal Victoria Hospital, of Montreal, on November 22, for a complete physical examination. This showed chronically infected tonsils and recession of the lower mandible. The liver and spleen were within the normal limits, and the cardiovascular system was normal. Examination of the respiratory system revealed showers of fine moist crackles, which did not clear when the patient coughed. The abdomen was soft, but there were no areas of tenderness and no abnormal masses. The blood count showed no abnormalities; the calcium and phosphorus contents of the blood were normal. The Wassermann tests of the blood and the cerebrospinal fluid were negative. The urine showed a few hyaline casts, and epithelial cells; albumin and a few white corpuscles were present. Roentgen examination gave negative results, as did examination of the pelvis and of the nervous system.

Examination of the Eyes.—Right Eye: The lids were normal and free from signs of irritation. A scar of the cornea extended from the nasal side to and over the greater part of the pupillary area, the result of phlyctenular disease in childhood. The pupil (4 mm. in diameter) reacted to light and in accommodation; the media were clear. Vision was counting fingers uncertainly at 1 foot (30.5 cm.). The field of vision could not be taken on account of the reduced sight, but on rough tests there seemed to be some defect in the nasal field. The ocular movements were normal; the tension was normal, and the fundus showed no changes.

Left Eye: The cornea was clear; the pupil was circular and active. Vision was 6/6. The field of vision was full; the fundus was normal, and ocular movements were normal.

Subsequent Examinations.—The patient was discharged from the hospital on November 28 but was readmitted on December 11, at which time the sole complaint was failing sight in the left eye, which had developed within the course of an hour or so.

Examination of the left eye showed the pupil to be dilated and inactive. Vision was perception of movements of the hand only. There was an apparent defect in the central field, as shown with tests with very large objects. The tension and the fundus were normal. Examination of the right eye showed the pupil to be dilated and inactive. Vision was counting fingers at 6 inches (15.2 cm.); the fundus was normal.

The following examinations gave negative findings: neurologic examination, dental examination, pelvic examination, tests of the cerebrospinal fluid, the Wassermann test of the blood, and roentgen examination of the skull. The heart and lungs were as previously reported. The blood count revealed 4,020,000 red cells, 3,650 white cells and 80 per cent hemoglobin. There were leukopenia, neutropenia, more immature forms of polymorphonuclears than mature forms and relative monocytosis. The agranulocytic character of the blood picture, the high lymphocyte count (relative) and monocyte count and the shift of the hemogram to the left demonstrated a marked myelotoxic process. The total number of the white cells was diminished.

Course.—During the patient's stay in the hospital there was a variation in her vision. The sight of the right eye ranged from counting fingers at 6 feet to 20/200. Then it declined to counting fingers at 4 feet (121.9 cm.). The vision of the left eye deteriorated from counting fingers to perception of movements of the hand. Then it decreased until there was no perception of light and remained so until the patient's death. The patient was discharged from the hospital on Jan. 28, 1933. The diagnosis was retrobulbar neuritis and secondary atrophy of the optic nerve.

On April 5 she was admitted to the neurologic service of the Royal Victoria Hospital under Dr. Russell on account of failing vision, a feeling of epigastric tightness and burning pain and pressure in the chest. She gave the following history:

About two weeks after leaving the hospital a burning pain suddenly occurred one night in the entire right leg; the feeling was as though the entire leg had something very hot applied to it. This feeling lasted a few days and then passed. A few days later a peculiar feeling of extreme tightness accompanied by a burning pain occurred across the upper part of the abdomen. While this burning pain was present the patient could not endure to have the bedclothes or other materials touch the skin of the affected regions. On a few occasions she noticed that when she touched the leg a peculiar sensation passed through it, as though an electric shock was being felt. A week or so after the pain first appeared she noticed that when she scratched the leg there was no sensation. The area of loss of sensation had progressed upward until it reached about the level of the fourth rib, anteriorly. Just before her admission to the hospital she became very constipated; she attributed this to the fact that she could not produce any pressure in the abdomen with the muscles.

Neurologic Examination.—A neurologic examination by Dr. Young revealed evidence of a lesion of the optic nerves and of the spinal cord. The other cranial nerves were normal. The mental status and speech were normal. There was no abnormality, subjective or objective, of the upper extremities, whereas the lower extremities, especially the right leg, were very weak. Paralysis of the right leg

was almost complete; the patient was capable of only slight movement of the foot and toes. Movement of the left leg was practically full but was weak. The tone was slightly increased in the right leg. No atrophy and no fibrillation were present. Coordination could not be tested accurately.

A lesion at the level of the fifth dorsal segment of the spinal cord on the right side was revealed by sensory examination. Touch was identified each time with possibly some impairment of the fifth lumbar segment on the right. There was loss of the sense of pain on the left side up to the seventh dorsal segment, just at the lower crease of the breast, where pain was felt as extreme. The sense of pain was impaired on the right over an area corresponding to the third to the tenth dorsal segment behind, and the fourth to the seventh dorsal segment in front. The areas of the disturbance of the sense of heat and cold corresponded to those of disturbance of the sense of pain. The sense of vibration was impaired in the right leg, and vibration was identified in the left leg. It was felt better at the sixth or seventh dorsal segment below. The sense of position was impaired in the right toe. The test with figure writing was not reliable. The cerebrospinal fluid showed an increased amount of protein (Pandy test, plus) and 40 lymphocytes per cubic centimeter. The Wassermann reaction was negative. This suggested Brown-Séquard's paralysis of the fifth dorsal segment on the right, and a diagnosis of encephalomyelitis was offered.

Final Examination of the Eyes and Subsequent Course.—The optic disks were pale and atrophic. Total blindness was present in the left eye. The patient counted fingers with the right eye at 8 inches (20 cm.).

On June 9 the patient asked to be discharged from the Royal Victoria Hospital with a view to going to New York for consultation, where she was admitted to the Mount Sinai Hospital on July 2. While there she contracted bronchopneumonia, and died on July 10.

Report of Postmortem Examination.—A postmortem examination confirmed the clinical findings of encephalomyelitis. The report of the postmortem examination from the Mount Sinai Hospital is as follows:

Coronal sections of the brain through the optic chiasm and tracts revealed on microscopic examination deposits of fat in the optic tracts and some demyelination through the chiasm. Many of the smaller vessels in this and other regions showed dilated perivascular spaces and cellular infiltrations. The spinal cord revealed patchy areas of demyelination and deposits of fat in various tracts at different levels. There was mild cellular infiltration of the meninges. On gross examination the dura was normal. The subarachnoid space was increased and was distended with clear fluid. The brain was softer than normal and injected. The convolutions were prominent, particularly in the region of the sylvian fissure. The sulci were correspondingly deepened. The arteries showed a mild degree of atherosclerosis. The ears and sinuses were normal. The eyes were removed (no report was made). The spinal cord was softer than normal and somewhat compressed along its entire length anteroposteriorly. Section of the brain revealed no alterations visible to the naked eye. Section of the spinal cord showed marked softening and disorganization of the mid-dorsal segments, with reduction in size of the upper dorsal segments. No other changes were seen with the naked eye.

The cause of death was acute encephalomyelitis of the brain and spinal cord, subacute meningo-encephalomyelitis and bronchopneumonia of the lower lobes of both lungs. There were fibrous pleural (in the right lung) and pleuropericardial adhesions; hydrothorax of the left lung; total adherent pericardium; necrotizing

cystitis; hydro-ureters and hydronephrosis (slight); ascending pyelonephritis; parenchymatous degeneration of the heart and liver; acute infectious splenic swelling, and scars of an old operation—cholecystectomy.

SUMMARY

Optic encephalomyelitis occurred in an otherwise healthy woman 34 years of age. There were rapid loss of vision of the left eye, with no recovery, and loss of vision of the right eye, with partial recovery. Paralysis of the right leg and of the abdominal muscles followed about four months after the onset of the disease in the optic pathway. The feature of special interest in this case is the long interval (four months) during which the course of the ocular phenomena remained obscure until the definite signs presented themselves in the spinal cord and thus completed the picture of optic encephalomyelitis. A further interesting finding was marked leukopenia, which was present during the entire course of the illness.

The Mount Sinai Hospital authorities furnished the report on the autopsy.

SCLEROMALACIA PERFORANS

REPORT OF A CASE

SAMUEL P. OAST, M.D.

NEW YORK

In the January 1934 issue of the ARCHIVES Dr. J. van der Hoeve,¹ of the university of Leyden, published a report of a series of four cases under the title of scleromalacia perforans. During the course of this report he stated that so far he had been unable to find any published account describing a similar condition and therefore concluded that his cases were the first recorded instances of a condition which he described elsewhere as follows:

A small slightly elevated inflammatory nodule appeared under the conjunctiva at a distance of about 10 mm. from the limbus. This nodule underwent necrosis at the center, discharged a yellowish cheesy material and then disappeared, leaving the deep blue color of the uvea in evidence beneath. This process repeated itself and showed a tendency to travel around the circumference of the eye in ring fashion and exhibited no disposition to involve the cornea or the anterior part of the uvea.

The case I am reporting appears to fit into this picture closely.

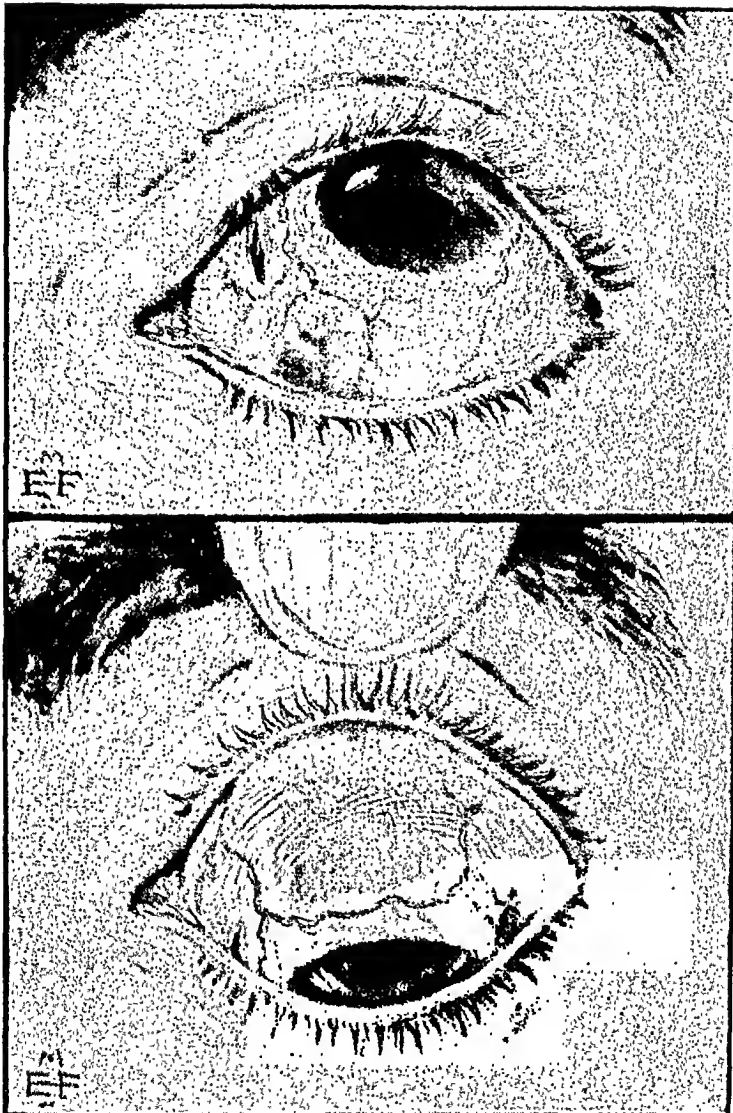
The patient was a man from Dr. Shine's clinic at the New York Eye and Ear Infirmary, 76 years of age, of robust appearance, who gave no history of previous serious illness, and did not recall ever having had any symptoms suggestive of rheumatism or arthritic involvement. I should like to emphasize this fact particularly, because in three of the four cases which van der Hoeve reported polyarthritis was a conspicuous accompaniment of the scleral picture.

The patient was first seen in the clinic about two years ago, at which time he came because of having been struck on the eye now affected (the left eye) by a chip while chopping wood. This blow was evidently severe, not judged so much by the external evidence (for all that could be found was a small laceration of the conjunctiva from 4 to 5 mm. long and just temporal to the limbus) but because of an extensive hemorrhage into the vitreous, reducing vision to perception of movements of the hand and precluding all efforts at examination of the fundus. It was thought that the sclera had not been perforated on this occasion because the tension remained normal and a roentgenogram taken to detect an intra-ocular foreign body showed no abnormality.

A few weeks after this injury the eye became quiet to all appearances. However, it was three months before the vitreous cleared enough to permit satisfactory examination of the fundus, when it was found to present no important changes. By this time visual acuity had returned to 20/30 without a correcting lens.

Read before the Section of Ophthalmology of the New York Academy of Medicine, Jan. 18, 1937.

1. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. **11**:111 (Jan.) 1934.



Scleromalacia perforans.

The eye remained quiet until about eight months ago (fifteen months after the injury which prompted the patient's first visit to the clinic), when there appeared, at about from 6 to 7 mm. from the limbus and just over the point of insertion of the superior rectus muscles, an inflammatory nodule approximately 5 mm. in diameter. This nodule was slightly painful and red, with a yellowish necrotic center, and appeared to be deeply seated under the conjunctiva. It was thought to be an abscess and was accordingly incised, but no pus was found, only caseous material being evacuated. After a few weeks this nodule had disappeared, and it could be seen that the underlying sclera had been virtually destroyed, leaving the bluish uvea in plain view beneath, except for a thin covering of translucent cicatricial tissue. While the original nodule was in the process of resorption a similar nodule appeared a short distance from the first, and this cycle has been repeated consecutively, the lesions traveling counterclockwise, until now fully two thirds of the eye's circumference has been traversed.

There has been a tendency for these foci to coalesce in places, leaving rather large areas where loss of scleral substance may be noted. The cornea has remained clear throughout, and there has been no sign of active involvement of the anterior part of the uvea. However, the opacities in the vitreous have increased so that no clear view of the fundus may be obtained at the present time. Vision is now reduced to the ability to see movements of the hand only, presumably because of the opacities in the vitreous.

I believe this to be a very rare condition, for, aside from the report of van der Hoeve's four cases, I have been unable to find any other mention of the condition in the "Index Medicus" or the "Quarterly Cumulative Index," though possibly it has been reported under another name. Most of my colleagues to whom I have shown the patient have never seen its counterpart before.

As van der Hoeve gave no suggestion as to the etiology of the condition in his cases, my associates and I naturally became interested in the condition from that standpoint. Because of the apparently low grade inflammatory process with termination in the formation of caseous material, it immediately suggested tuberculosis. Therefore on three separate occasions we inoculated an albino rabbit with caseous material obtained from the foci, twice in the anterior chamber and once under the conjunctiva, but without producing any comparable pathologic process or, for that matter, any noteworthy inflammatory reaction.

Previously the usual laboratory examinations, including smears and cultures, were done; these yielded no organisms aside from the normal inhabitants of the conjunctival sac.

The Wassermann reaction was negative, and the blood sugar content was 100 mg. per hundred cubic centimeters. General physical examination revealed nothing of moment. A search for distant foci, including roentgen examination of the teeth, nose and throat, showed nothing unusual, and a dark field examination for *Spirochaeta pallida* gave negative results. A biopsy was done and showed only granulation tissue and cellular debris. No tuberculin tests were carried out, because

it was thought that the negative results obtained in the repeated inoculation of rabbits had excluded direct tuberculous infection, and tuberculous allergy was not seriously considered because of the appearance and behavior of the lesions. Perhaps these tests should have been carried out also.

At no time was any appreciable involvement of preauricular or any general febrile reaction detected. The appearance of the lesions, however, did suggest a similarity to those found in squirrel plague or in conjunctivitis necroticans, and with this in mind my associates and I, with the help of Mr. Burchell, tried to induce a growth of fungi on glycerin agar (having previously tried the ordinary stock mediums). We also made efforts to produce a growth in the absence of oxygen, but our efforts in these directions, as in others, proved unavailing, and we know no more about the etiology now than when we started.

The term scleromalacia is suggestive of some analogy between this condition and keratomalacia. Naturally, one thinks at once of vitamin deficiency as a producing factor, and the condition doubtless will bear more investigation along these lines. However, if this were the case one would expect the cornea or the fellow eye to become involved, and I should like to emphasize at this point that neither eye has presented any clinical evidence of pathologic changes at any time within the period of our observations.

The obvious predilection of the process for the sclera and the freedom enjoyed by the usually more vulnerable structures, the uvea and cornea, in spite of their proximity, suggest to some (Wood and others²) a disturbance of the calcium metabolism, and it would not have been amiss to have a determination of the blood calcium content made during the active stage of the disease, but it did not occur to me at that time.

I cannot help but believe that some connection exists between this condition and the patient's previous injury, but in what manner or to what extent is extremely problematic.

At no time has any rise of the intra-ocular tension been observed, but even with normal tension it is difficult to explain why there has been no tendency to staphyloma when the uvea appears to be almost the sole retaining tunic.

Our treatment has been symptomatic and varied. The one thing which seems to have exerted a beneficial influence was irradiation with ultraviolet rays administered by means of a Birch-Hirschfeld carbon lamp with a uviol filter. After the first few exposures the eye became appreciably quieter, and the pain lessened. There has been continued

2. Wood, D. J.: Calcium Deficiency in the Blood with Reference to Spring Catarrh and Malignant Myopia, *Brit. J. Ophth.* **11**:224 (May) 1927.

improvement ever since, the eye at present being practically free from inflammatory symptoms. Just how much the ultraviolet therapy had to do with the improvement it is hard to say, but no doubt exists in my mind or in that of the patient that the course of the disease took a definite change for the better with the beginning of these exposures.

Van der Hoeve, in discussing the condition, stated that he had chosen the name scleromalacia in preference to scleritis necroticans, as had been suggested, because of the relative freedom from inflammatory signs, but in the case reported here there has never been any question in the minds of those who saw the condition in its more active stages that a true inflammatory reaction, though relatively low grade, was in progress. At any rate, the picture would not correspond clinically to my understanding of primary degeneration.

It looked, at one time, as though we were going to secure a pathologic specimen, which perhaps would have shed some light on the etiology, but, owing to the efficacy of the ultraviolet rays or, perhaps, to the termination of a natural course, that possibility has become remote.

ORGANIZATION OF THE DEPARTMENT OF OPHTHALMOLOGY OF THE LONG ISLAND COLLEGE OF MEDICINE

JOHN N. EVANS, M.D.

BROOKLYN

The department of ophthalmology of the Long Island College of Medicine¹ has inaugurated what is probably a new method for the training of young men for the specialty of ophthalmology.

In order to appreciate this method, which may be called supplementary fellowship, it is necessary that the general plan of organization of the department be outlined.

The clinic and hospital, though theoretically separate, are for the most part staffed by the same physicians, and in practice are nearly identical. All the members of the staff aid in teaching undergraduates, though only a few hold teaching appointments.

There are a director and an assistant director of the dispensary, who are also the ophthalmologist in chief and the associate ophthalmologist, respectively, to the hospital. They represent the college as professor and clinical professor, direct policies and maintain coordination. The work in the dispensary is arranged as follows: Division A serves Monday, Wednesday and Friday, and division B serves Tuesday, Thursday and Saturday.

Each division has a chief of clinic, who is directly responsible for the management of the cases in his division. Each of the remaining members (sixteen) of the staff has a maximum of six cases assigned to him each day in the clinic (three new cases and three old cases). He studies these cases carefully, any necessary advice or assistance being given by the chief of clinic. The interns, residents and fellows are assigned cases the same as other members of the staff of the dispensary. Should the case be referred to a special consultation clinic (the clinic for patients with glaucoma, the clinic for patients with squint, the clinic for perimetry, the general medical clinic or the neurologic clinic) or should the patient be admitted to the hospital for operation or study, his physician must keep in contact with him and must be present with him on every possible occasion. It is only through such

1. The Long Island College Hospital and the dispensary work in conjunction with the Long Island College of Medicine.

personal study and follow up that the younger physicians can be trained in the proper management of cases in private practice.

Members are first trained in cadaver surgery, then in minor surgery and finally in major surgery. As their aptitude becomes more evident they are eligible for promotion. Seniority of service alone plays a minor part in promotion. Aptitude and genuine interest in ophthalmology are the determining evidence of fitness for advancement.

The internships are open primarily to young physicians who plan to practice in Brooklyn, who have been graduated from a grade A school and who have served an internship in a general hospital—in a medical or rotating service in an institution approved by the American Medical Association. Applicants are accepted after a personal interview by the ophthalmologist in chief. There are no formal examinations.

The internship in ophthalmology (it is termed a residency in ophthalmology by the Council on Medical Education and Hospitals of the American Medical Association) is for one year and follows a definite program of hourly duties and instruction. Assignments are listed in a formal program arranged for each month, and detailed duties, obligations and methods are set forth in the constantly available manual of the hospital.

The residency in ophthalmology is open to the intern after a satisfactory service in this institution (or its equivalent, as determined by the ophthalmologist in chief). The residency is for one year and is likewise open to those who anticipate practicing in Brooklyn. The resident follows a program of duties and instruction similar to that which is followed by the intern.

The fellowships—two in number at present—are open only to young men who have completed the internship and residency just outlined (or its equivalent, in the opinion of the ophthalmologist in chief). They must have opened their private offices in Brooklyn and must maintain active practice in Brooklyn in ophthalmology at least three days a week. The alternate three days are devoted to their fellowship work. The stipend for the fellowship is (at present) \$1,200 a year, but this amount may be varied at the discretion of the ophthalmologist in chief.

The primary purpose of the fellowship is to round out the training of these young physicians so as to prepare them adequately for the examinations of the American Board of Ophthalmology. In some instances it may be advisable to limit a fellowship to one year; in other instances it may be necessary to continue the fellowship two, or even three, years. The fellow assists in the instruction of interns, residents and undergraduate medical students and receives instruction and training by means of an assignment of hourly duties as arranged by a scheduled

monthly program. He agrees to accept appointment to the staff of the dispensary on the completion of his fellowship. In this way physicians of the school's own training staff the dispensary and, later, the hospital.

Besides the obviously continuous duties as set forth in the sample program, the fellows, residents and interns receive progressive instruction each month between 10 and 12 a. m. and between 3:30 and 5 p. m. For instance, courses in anatomy and embryology are conducted by the department of anatomy of the college; courses in pathology of the eye are given under the auspices of the department of pathology of the

*Program for Fellows and Residents, November 1936**

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
8-9	Rounds	Cheek records	Cheek records	Rounds	Cheek records	Cheek records	Free
9-10	Refraction clinic	Rounds	Rounds	Refraction clinic	Rounds	Rounds	Rounds
10-11	Refraction clinic	Anatomy	Anatomy	Refraction clinic	Anatomy	Anatomy	Free
11-12	Refraction clinic	Anatomy	Anatomy	Refraction clinic	Anatomy	Anatomy	Free
12-1	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch
1-2	Clinic	Clinic	Clinic	Clinic	Clinic	Clinic	Free
2-3	Clinic	Clinic	Clinic	Clinic	Clinic	Clinic	Meet relatives
3-4	Glaucoma clinic	Operating	Staff rounds	Glaucoma clinic	Operating	Staff rounds	Free
4-5	Staff rounds; instruction in refraction	Staff rounds; instruction in refraction	Instruction in refraction	Staff rounds; instruction in refraction	Staff rounds; instruction in refraction	Open	Free

* Time for attendance at neighboring seminars, courses and society meetings is arranged.

hospital, and instruction in optics is conducted by the departmental optician. There are special courses under the department of physiology, the department of neurology and the department of medicine, and the young physicians are required to take part in, or initiate, research work. Special courses are given in various branches of ophthalmology, not only during the hours spent at the clinic but also regularly from 3:30 to 5 p. m. throughout the year.

Examinations and quizzes, assignments from texts and the reading of periodical literature are requisites. The fellows must write a preliminary report at the end of the first six months and a thesis at the end of the first year.

Clinical Notes

CONGENITAL NYSTAGMUS

L. PELLMAN GLOVER, M.D., ALTOONA, PA.

The recent report of Cox¹ on congenital head nodding and nystagmus has led me to question more closely in cases of nystagmus the history of other members of the family in an attempt to trace a hereditary element, even though no head nodding was present. I was extremely fortunate in obtaining the genealogy of four generations through the efforts of the mother of one of my patients with nystagmus. The history may be relied on, as the mother is very intelligent and closely questioned other members of the family.

She volunteered the information that the women never had the trouble, but seemed to transmit it to the males. She also mentioned that by some coincidence the fourth born, if a male, had the trouble

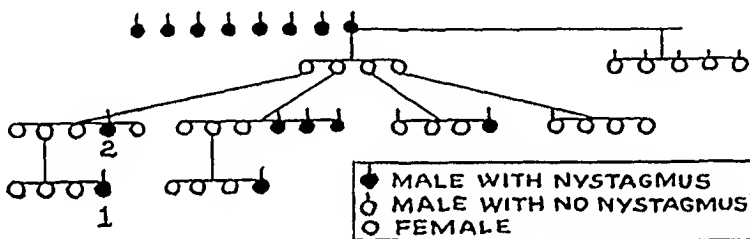


Diagram of genealogy of four generations of the family showing hereditary nystagmus.

which the earlier members escaped. To quote from her own statement, "My grandfather's eyes were crossed and roving. He had seven brothers, all of whom had similar eye trouble. They must have made a pretty picture at the table. Grandfather had five sons and four daughters who were normal. Each daughter, with one exception, had one or more children with roving eyes. Some had nodding of the head or held the head back. The eyes always roved, but the nodding or holding the head back usually stopped when they grew older."

The family tree is shown in the diagram. The mother of the member numbered 1 gave the information. Only the members numbered 1 and 2 have been examined by me. The findings in their cases are reported.

REPORT OF CASES

CASE 1.—The patient was C. B., aged 4 years. The family noticed that both eyes showed a rotatory nystagmus since birth. As soon as the child began to

1. Cox, Ronald A.: Congenital Head-Nodding and Nystagmus, Arch. Ophth. 15:1032 (June) 1936.

walk the parents noticed that he held his head down and looked up in order to see straight ahead. There was no injury sustained at birth.

Examination showed an apparently normal and mentally alert child. He held his head down and looked up to see, as a person would over the top of a pair of glasses. An equal rotatory nystagmus was present, but there was no squint. Aside from this all the findings were normal. On refraction a -1.00 sphere was ordered for the right eye and a -1.25 sphere for the left. Examination five months later, in November 1936, showed an improvement in the nystagmus and in the tilting of the head.

CASE 2.—H. W., aged 17, an uncle of the first patient, was first seen in May 1934. He had been wearing glasses for five years. Nystagmus had been present from birth, and the right eye had always turned in.

Examination showed normal holding of the head. There was a rotatory nystagmus of both eyes, with a convergent squint of 35 degrees of the right eye. The pupils were dilated and reacted poorly to light. Both optic nerves showed a slight gray atrophy. Refraction with the eyes under the influence of homatropine showed vision in the right eye of 6/30 with a $+1.50$ sphere $+3.25$ cylinder, axis 90, and the patient could read Jaeger's test type 10. Vision in the left eye was 6/21 $+$ with a $+1.50$ sphere $+3.50$ cylinder, axis 105, and the patient could read Jaeger's test type 10.

The patient was instructed to carry out fusion exercises and in two months was able to fuse satisfactorily with the eyes straight. There was no change in the nystagmus. When he was last seen, in August 1935, the condition had remained stationary. The patient has always been in perfect health and is mentally alert.

COMMENT

While in the cases just reported the patients did not show the typical head nodding, the first patient evidently held the head back to compensate for the nystagmus. I feel that the condition just described would come under Cox's classification of head nodding and nystagmus. Unfortunately, other members of the family could not be studied, as they live too far away. In any event, the family history of hereditary nystagmus is evident. The unusual feature is the apparent transmission through the females, who did not have the disease, to the males, who were susceptible.

A NEW HOLSTER FOR THE OPHTHALMIC WARD AND CLINIC

SAMUEL PELUSE, M.D., CHICAGO

The ophthalmoscope holster here described was designed in the department of ophthalmology of the Cook County Hospital, where it is now in daily use by the interns and resident physicians.

Its practical features have been fully attested to by those who have used it in the routine examination of patients in the wards and clinics.

The holster eliminates the loss of time and patience attendant on the misplacing of the ophthalmoscope and other accessories, such as a pencil or pen, a rule, a retinoscope or a pocket flash-light during rounds of the wards. It dispenses with the disarrayed and frayed pockets encountered when the ophthalmoscope is carried in the usual manner on one's person. The continual hazard of "hooking the chair" or other objects when carrying the ophthalmoscope in the rear pocket

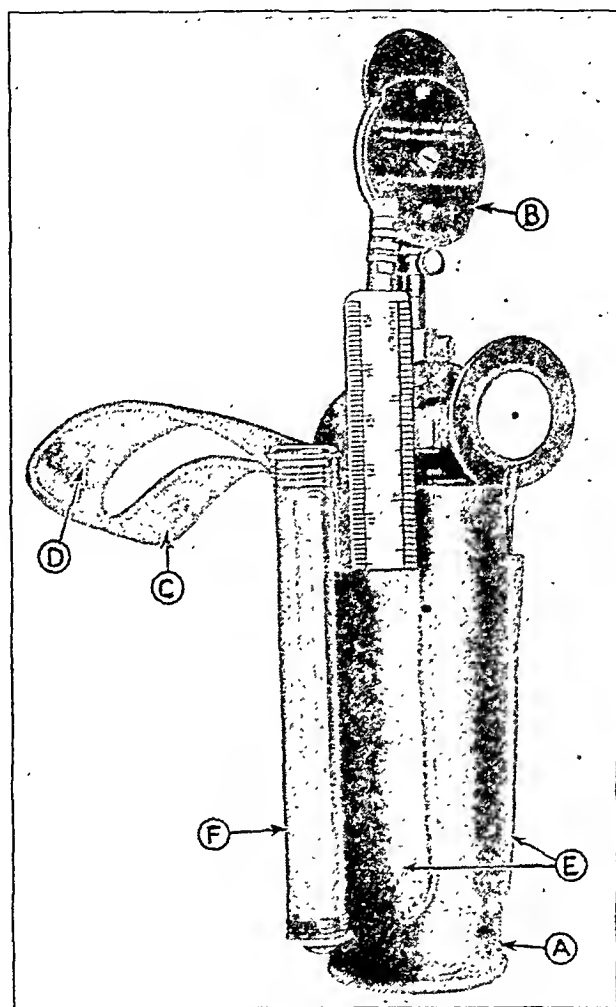


Fig. 1.—The Peluse ophthalmoscope holster in detail.

of the trousers is eliminated. The danger of damage to the ophthalmoscope by accidentally dropping it on the floor is done away with.

During the short period that it has been used (the past six months) the cost of maintenance of the ophthalmoscopes, as verified by the purchasing department, has dropped to a minimum.

The device (fig. 1) is made of leather and weighs 3 ounces (85 Gm.). It consists of a sleeve (A) 5 inches (12.7 cm.) in length and $1\frac{3}{4}$ inches (4.5 cm.) in diameter, open at the top and closed at the bottom for ensheathing the battery case of the ophthalmoscope (B). This sleeve is supported by a strap (C) made of stiff leather $1\frac{1}{2}$ inches

(3.8 cm.) wide and 11 inches (28 cm.) long which is stitched to the back and fitted with a snap catch (*D*) for straddling the wearer's belt.

On each side, on the outside of the sleeve is a pocket (*E*) 1 inch (2.5 cm.) wide and 3 and $\frac{3}{4}$ inches (9.5 cm.) long, used as a receptacle for the retinoscope, pencil and flexible rule. The pocket flash-light (*F*), as well as the pen (if it is used), may be supported by slipping the clip over the free edge of the sleeve or over the edge of one of the side pockets.

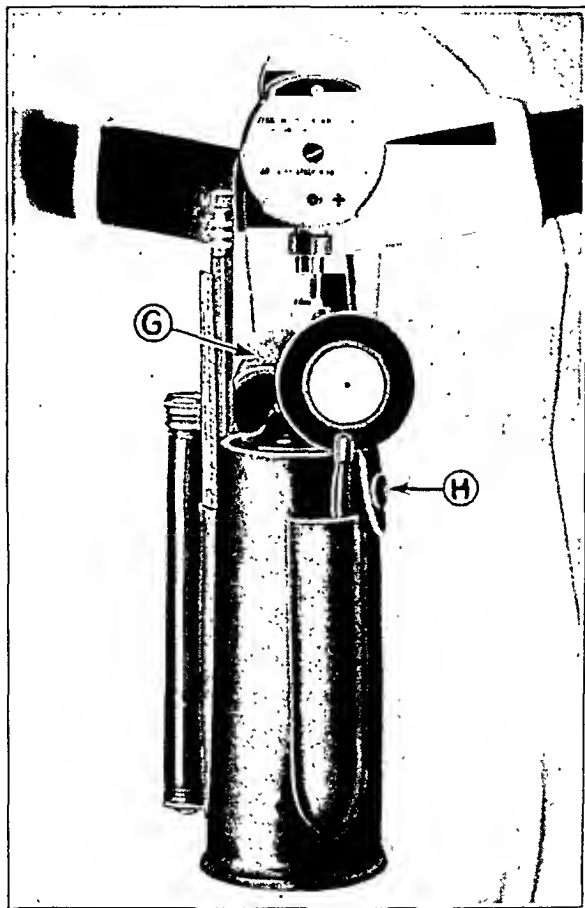


Fig. 2.—The Peluse ophthalmoscope holster strapped to the wearer's belt.

A thin strap of leather (*G*) $\frac{1}{2}$ inch (1.2 cm.) wide and 5 inches (12.7 cm.) long (fig. 2) is sewed to the supporting strap and allowed to curve over the shank of the ophthalmoscope; it is provided with a snap catch (*H*) on the end to prevent the ophthalmoscope from falling out of the sleeve.

The holster snaps readily over the wearer's belt and may be worn conveniently, as illustrated in figure 2.

This holster is a product of the Peluse Specialties, 105 South Jefferson Street, Chicago.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

PHOTOGRAPHY IN OPHTHALMOLOGY

ARTHUR J. BEDELL, M.D.

ALBANY, N. Y.

The study of art, particularly of the paintings of the thirteenth and fourteenth centuries, shows that the artists' concepts of anatomy, from the present day standpoint, at least, were crude and impossible. Even today the most skilful portrait painter combines his interpretive insight with his manipulative skill. So it must necessarily follow that the old statement "Things are to the eye that sees them as to that eye they seem to be" is still true. It is one thing to have a desire to make a picture of beauty and another to get a faithful reproduction of the details. If one wishes scientific accuracy a photograph is necessary.

Photography has advanced so far that it needs no defense, but occasionally an explanation of how to use it tends to widen its field of application and make the results more presentable and more worthwhile. This paper is designed to draw attention to some of the practical rules of photography and the particular reasons for using different cameras in the special field of ophthalmology. It is a presentation of the methods I use.

When certain underlying basic principles are understood, all photography becomes comparatively easy and simply a matter of technical control while using the subject, manipulating the camera, adjusting the source of light, selecting the sensitive plate, developing it and, finally, printing from the negative.

I have found it easier to take a satisfactory photograph of the face or of the external portion of the eye when the patient is seated in a comfortable chair with the head placed against a white silk screen, which is not held taut but allowed to hang in loose folds. The illumination comes from two no. 2 Mazda photo-flood lamps in the ordinary Eastman Kodaflector, placed 3 feet (91.4 cm.) from the subject in a position to bring the lights and shadows with the desired contrast on the patient. Almost any camera may be used for this purpose, but it must be on a tripod, because the time of exposure is too great for ordinary steadiness of hand. I use the large Graflex camera, the National Graflex camera or the Leica camera. I prefer the large Graflex camera, because with it the subject is visualized up to the instant the picture is taken.

Most of my pictures of the external portion of the eye are made with the camera 3 feet from the patient. A Weston light meter is used to determine the size of the diaphragm opening and the time of exposure. The correct use of this instrument will avoid many disappointments caused by faulty exposure.

To focus the patient's face accurately the diaphragm is opened to its maximum size and then closed to $1/16$ or $1/32$, and when all is in readiness the plate is exposed for the predetermined proper length of time, which is with the former two-fifths second and with the latter one and one-half seconds, if a film with a 16 value, such as Verichrome, is used. Other types of film call for different times of exposure. In the ordinary case, speed is not essential for this kind of work, and therefore any of the standard double-coated films or plates can be used.

When one is working with a miniature camera, the position of the patient, the light and the camera are arranged in a manner similar to that for the larger apparatus. The built-in focusing mechanism enables one to adjust the camera to the selected distance. Great definition of depth is secured with the Leica instrument.

There are two ways of taking stereoscopic photographs with this camera. By one method the first picture is made in the usual manner; the camera is then moved a predetermined distance on the stereo slide bar, and the second picture is made under the identical conditions which pertained to the first. The other way is to use the stereoly attachment, by means of which the stereoscopic pictures are made with one exposure. With this method photographs are taken in a few minutes and supply an invaluable record of tumors, exophthalmos and deviations in the facial contour.

My clearest pictures of the external portion of the eye are made with the large Graflex camera, a $1/32$ diaphragm opening for a one and one-half minute exposure of a Verichrome film being used. Because of the ease of handling, I prefer the film, but of course a plate can be used, and this gives ideal results.

A longer exposure is necessary to take a colored photograph. The light must be correct and the focus and exposure perfect, for it is essential that everything be accurate if one is to have clear pictures. More attention to detail is necessary when using color plates than when taking photographs in black and white. When a filter is used it should be placed before the photographing lens rather than over the source of light. This, I believe, is important.

When changing from one type of film to another it is necessary to remember the different time of exposure, and one should adjust the diaphragm accordingly. The proper exposure should always be calcu-

lated by using the light meter, for even after long experience I find that the complexion of the patient has much to do with the definition secured on the film.

For photographs of the lids, conjunctiva and anterior segment of the eye, I use one of three cameras, the Bausch and Lomb Stereocamera, the Zeiss Iris stereoscopic camera and the Drüner stereoscopic camera. Each has its field of use and its limitations.

The Bausch and Lomb Stereocamera is easy to operate. The patient's head is held in a rigid frame with a chin cup and a forehead rest. The light comes from two ordinary motor headlight bulbs projected through a lens system, which is an integral part of the camera, which in turn is mounted on a rigid base with adjusting screws. The part to be photographed is focused on the ground glass plate, and its position is noted through the focusing tube. The plate holder is placed in proper position, and the time of exposure is determined. The object is kept under control by observation through the focusing tube up to the time of exposure. There is no adjustable diaphragm with this camera, so that it is easy to get a clear stereoscopic picture by the exposure of ordinary orthochromatic plates for one twenty-fifth second. A filter is necessary when one is using the Dufay plates but not when using the Agfa color plates. For the latter a one-half second exposure gives the best results.

Infra-red plates should be used when the cornea is opaque. An exposure of one second gives surprisingly excellent pictures.

The Zeiss stereoscopic camera has a somewhat limited range of usefulness. It is well adapted to taking photographs of the eyelids, conjunctiva, eyeball and iris. It is a compact, excellent instrument with a built-in light, a tube for observation and a diaphragm control, but, unfortunately, it is not made for the present day color plates. The patient is held firmly against a forehead rest and a chin rest, and the plate is exposed in the usual way.

With the Drüner stereoscopic camera the object is focused on the ground glass plates, with control by observation through a telescopic focusing tube. The light is not a part of the instrument and must be supplied from an external source. The diaphragm opening is fixed, and the time cannot be regulated, so that to get a clear photograph it is necessary to have an intense illumination. I use a carbon arc lamp on an adjustable standard. The advantage of the instrument is that one can photograph all the parts of the eye back to the posterior portion of the capsule of the lens. Therefore, this is the camera of choice in photographing corneal changes, including endothelial deposits, altera-

tions of the iris and abnormalities of the lens. It is much more difficult to use than the other cameras, but no other gives such a wide range with considerable magnification of the object.

PHOTOGRAPHY OF MOVING OBJECTS

For the permanent record of defects of ocular motility, especially lesions of muscles, nothing serves as well as a clear moving picture. Changes can be portrayed just as well in the plain black and white film as in the color film. Kodachrome A film sensitized for photoflood lights gives beautiful colored pictures, and is both practical and inexpensive. The camera and the flood lights are placed so as to illuminate the object clearly and give the shadows their proper value. The size of the diaphragm opening is regulated by the reading on the photometer. It is not hard to take good moving pictures if the field is kept well lighted and in focus. The surgeon with extensive clinical opportunities and expert operative skill who wishes to record his observations should use the moving picture camera in daily practice.

PHOTOGRAPHS OF THE FUNDUS OCULI

The problems confronting the photographer of the fundus are by no means insuperable. If due attention is paid to the necessary details any one can make excellent pictures. Even at the risk of being elementary, it seems advisable to deal with this part of the technic step by step rather than by giving a general outline.

Preparation of the Patient.—The pupil must be dilated as widely as possible. This can be done with homatropine or eucatropine.

Adjustment of the Light.—The carbons of the arc lamp must be constantly adjusted. The direct current must be under complete control, 5 amperes and 110 volts being used. The carbon carrier must be placed so that the maximum brilliant illumination is secured. This is done by holding a card a few inches in front of the lens while moving the carrier to the site where the greatest efficiency results and locking it in that position. Many beginners experience difficulty by failing to heed this important recommendation.

Position of the Patient.—The patient's head is fitted into the forehead rest by changing the position of the chin cup by means of the adjusting screw which controls it and by raising or lowering the stool on which the patient sits. The head must be held rigid during the entire time of focusing and picture taking. I prefer to have an assistant keep it firmly in position.

Focus of the Light.—The band of light is partly focused on the side of the eye to be photographed. The other eye is fixed on the object held by the assistant or on the spot in a mechanical fixation device.

I prefer to use an assistant, for her hand can be moved by me as I desire to change the position of the eye. The camera light is then turned into the pupillary area and focused as a narrow band on the cornea, preferably just above the inferior edge of the pupil.

The position of the light and the angle at which it enters the pupillary space will depend on the portion of the fundus to be photographed, and an effort should be made to keep the light in the place where the intensification of lights and shadows give the clearest view. The time of exposure is previously determined by the ophthalmoscopic examination of the fundus. The plate is placed in the holder, and the slide removed, while the photographer continues his observations through the ocular. When all is in readiness the mirror is released and the shutter automatically opened. My best pictures of the average fundi are taken in one-half second on a double-coated orthochromatic plate. For pale fundi the time is reduced, and for dark ones it is increased.

Stereoscopic Pictures, Colored Pictures and Infra-Red Photography.—When a stereoscopic picture is desired, the base of the camera is displaced 44 mm., and the process is repeated. It is essential—in fact, absolutely necessary—that the photographer remember the precise spot where the light from the carbon arc was placed in the fundus view and bring the eye to that identical position. After everything is adjusted the plate is exposed in the same manner as for the preceding picture.

Colored pictures are taken on Agfa plates, and instead of lengthening the time of exposure I increase the illumination, using $7\frac{1}{2}$ amperes and 110 volts, with direct current.

Infra-Red Photography.—For infra-red photography it is necessary to remove the fixed filter from the camera and put another one in its place. So far I have found little advantage in infra-red pictures of the fundus.

Colored fundus photographs are of increasing value, but as yet they cannot take the place of black and white photographs in showing details of the structure of blood vessels. They have an appeal to the general physician because he sees the fundus in a way similar to that to which he has become accustomed. The trained photographer also knows that the colored picture helps to locate some of the deeper retinal and choroidal lesions, particularly those which are overshadowed by a hazy retina. Colors are also desirable in papilledema, papillitis, retinal hemorrhages and exudates. My most beautiful color pictures are taken on Agfa plates without the interposition of a filter. Because of the construction of the fundus camera, it is a little difficult to obtain any great degree of success with filters.

As the camera has no diaphragm control the exposure must be regulated by changes in the intensity of the illumination and in the time of exposure.

Stereoscopic photography of the fundus with the stereoscopic camera has not proved practical. To secure a good picture with the instrument it is necessary to have a dilatation of the pupil of from 7 to 8 mm., and the practicing ophthalmologist appreciates that it is impossible to get this in several conditions, notably, hypertensive states.

A camera is on the market for the photographic reproduction of the corneal surface by means of placedo disk reflections. The latest camera is for photographing the angle of the anterior chamber, but as yet this has not been perfected in mechanical arrangement, and the results leave much to be desired.

Comment.—Every clinician should take photographs; how many photographs he takes and under what conditions depend on his personal interest. Every ophthalmologist should be competent to read a photograph of the fundus. The day has passed when the ophthalmologist can go on the witness stand and say that he is unable to interpret a picture of the fundus, for this is a frank admission of his lack of knowledge, understanding and progressiveness.

SUMMARY

A satisfactory picture depends on the selection and manipulation of the camera made for the particular purpose, the correct angle and intensity of the illumination and the proper film. The smaller the diaphragm opening, other factors being equal, the greater the depth of the picture; so an attempt should always be made to observe this rule.

Until the photographer has become skilled he should entrust the development of the film and the printing of the picture to a trained technician, preferably a commercial photographer, who has by training and adaptability proved his worth as a cooperative agent.

In answer to many requests I include the list of the films I use:

For photographs of external conditions, the Eastman Verichrome film.

For photographs showing details of the lens and cornea, Hammer Ortho Plates Special Non-Halation, D. C.

For plain motion pictures, the Eastman panchromatic film.

For color motion pictures, the Eastman Kodachrome "A" film.

For infra-red photographs, Eastman infra-red plates.

For black and white pictures of the fundus, Hammer Ortho Plates. Special Non-Halation, D. C.

For colored pictures of the fundus and for colored pictures of the external part of the eye, Agfa color plates.

Since the early part of March, I have been using Kodachrome A in a film carrier which I had made for the fundus camera. The photographs are the best colors I have ever had.

Correspondence

OPHTHALMOLOGIC NOMENCLATURE

To the Editor:—I venture to call the attention of the ophthalmologic bodies to the desirability of appointing a committee on ophthalmologic nomenclature. Such a committee should go into the subject thoroughly and standardize and simplify many of the ophthalmologic terms which are at present used loosely or incorrectly.

To mention one term, let prism diopter be considered. As every one knows, the unit and the term were coined by Mr. Charles F. Prentice. Both have long been adopted by all the American manufacturers, who for several decades have been grinding their prisms and calculating all decentrations of lenses in terms of the prism diopter. Still many published articles continue to speak of prisms in degrees, and many a prescription is written for a prism of, say, 2 degrees. It is safe to state that the prism used in the test was a prism of 2 prism diopters, and certainly the prism in the prescription will be a prism of 2 prism diopters, since prisms are no longer ground according to the degree system.

In the recent work on ocular muscles by Dr. Peters one finds prisms spoken of in terms of centrad. The centrad proposed by Dr. Dennett was never adopted in this country, and prisms are not ground on the centrad system. I believe that Dr. Peters meant prism diopters when he wrote centrad; yet the centrad and the prism diopter are different units. The former is based on an angular measurement; the latter, on a linear measurement. It is true that the actual effective difference between prisms made according to the three systems of degree, centrad and prism diopter is slight. Still, it is inaccurate to mix the terms indiscriminately.

The general adoption of a correct terminology for prisms would eliminate a frequent source of confusion in discussions of the measurements and treatment of squint. For instance, when in the literature a condition is reported as a squint of, say, 15 degrees, one frequently does not know whether that means 15 degrees of actual angular deviation as measured on a perimeter, a method which Dr. Peters favors, or 15 prism diopters as measured by the cover test with loose prisms, as practiced by Dr. White. If the word "degree" is never used to denote prism values, any reference to degrees would mean only one thing—angular deviation as measured on a perimeter or a similar instrument.

I believe the principal reason for the lag in the use of the term prism diopter is the awkwardness of the term as compared with the terms degree and centrad. Various attempts have been made to simplify the term prism diopter to prismopter, prisopter and similar substitutes. But these terms are still too clumsy. A committee on nomenclature could agree on a suitable term and have it generally adopted. Personally, I have frequently used the word "delta," which is the name of the Greek letter Δ , at present used as the symbol for prism diopter. This seems a suitable term and is as short and euphonious as "degree" or "centrad." Thus one would speak of a 1 delta prism, a 5 delta prism, etc.

Another desirable matter for such a committee to take up would be the advisability of describing the position of the prism by its apex rather than by its base. The image seen through a prism is in the direction of the apex, and a prism both for measuring and for relieving heterophoria is placed with its apex in the same direction as the deviation of the eye. It is much easier to correlate the position of the apex of the prism with the ocular deviations than it is to correlate that of the base, the latter correlation requiring inversion and some mental gymnastics. This is a waste of mental energy and hampers rapid and effective thinking.

The foregoing are only a few suggestions. There are many more terms which could be simplified and standardized by such a committee in the interests of clearer and more accurate thinking and writing.

JOSEPH I. PASCAL, M.D., New York.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Examination by the American Board of Ophthalmology.—The American Board of Ophthalmology announces that, in addition to the examination to be held in Philadelphia on June 7, 1937, another examination will be conducted in Chicago on October 9. All applications and case reports, in duplicate, must be filed at least sixty days before the date of examination. Further information may be secured from the secretary, Dr. John Green, 3720 Washington Boulevard, St. Louis.

SOCIETY NEWS

American Academy of Ophthalmology and Otolaryngology.—Announcement has been made by the American Academy of Ophthalmology and Otolaryngology that the meeting place of the 1937 convention has been changed from Detroit to the Palmer House, Chicago. The convention will be held from October 10 to 15.

French Ophthalmologic Society.—The One Hundred and Fiftieth Congress of the Société Française d'Ophthalmologie will take place in Paris, at the Faculté de Médecine, on June 28, 29 and 30.

Philadelphia County Medical Society, Eye Section.—The following program was presented at the scientific meeting of the Eye Section of the Philadelphia County Medical Society, held on March 4:

Presentation of clinical cases from the Wills Hospital, by Dr. Henry Irwin.

"Albinism from the Standpoint of Pigment Defects," by Dr. Howard Rome.

Discussion of albinism, by Dr. P. De Long.

"Further Studies in Dark Adaptation as a Clinical Test." by Dr. Jacob B. Feldman.

Discussion of dark adaptation, by Dr. Edmund B. Spaeth.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Aqueous Humor

THE AMOUNT OF OXALIC ACID IN THE AQUEOUS. O. BARATTA, Arch. di ottal. 43: 44 (Jan.) 1936.

Baratta followed Maugeri's method, employing 5 cc. portions of aqueous from the eye of the horse, ass, ox, calf, goat and sheep. For the smaller animals, the smaller amounts of aqueous obtainable were diluted with water to 5 cc. Eight to twelve samples were run for each species of animal. The amounts of oxalic acid found, in milligrams per hundred centimeters, were 7.8 in the horse, 7.2 in the ass, 9 in the sheep, 7.9 in the calf, 8.4 in the goat and 8.61 in the ox. The presence of oxalic acid in the human aqueous was determined, but the amount was not estimated.

S. R. GIFFORD.

Biochemistry

SULPHUR METABOLISM IN SENILE CATARACT. M. C. BOURNE and D. A. CAMPBELL, Brit. J. Ophth. 20: 684 (Dec.) 1936.

Bourne and Campbell state: "The results of this investigation do not reveal any difference in the urinary sulphur distribution in the cataractous patients as compared with the control cases. Although the individual variations are great, the results are in all cases similar, and in general follow the classical distribution of urinary sulphur first described by Folin (1905). That is, the largest fraction of the total sulphur is excreted as inorganic sulphate which varies with the amount of protein ingested; while the ethereal sulphate and neutral sulphur fractions remain more or less constant and independent of the total sulphur excretion. In none of the patients with senile cataract is any deviation from the normal to be observed in the behavior of any fraction. Therefore the results lend no support to the hypothesis that a disorder of sulphur metabolism might be the basis of the pathological changes in the lens in senile cataract; but the experiments are not sufficiently extensive or conclusive to rule out the suggestion completely. A detailed study of the neutral sulphur fraction, the composition of which even in normal individuals is not completely understood, might show the presence of some specific compound associated with senile cataract. The fact that no increased excretion of the ethereal sulfates was found in any of the cataractous patients in our series makes it unlikely that senile cataract is due to a toxic process. Blood glutathione estimations were done on a number of these patients; the results, which have already been published (Campbell, 1936), showed a normal blood glutathione in every case."

W. ZENTMAYER.

EFFECT OF ALPHA-DINITROPHENOL ON THE OXIDATION-REDUCTION POTENTIAL OF THE AQUEOUS AND THE LENS. J. NORDMANN and P. REISS, *Compt. rend. Soc. de biol.* 123: 233, 1936.

Attempts to produce lenticular opacities in the rabbit and the rat with dinitrophenol were unsuccessful. In normal rabbits the potential of the aqueous and the lens was found to be plus 143 millivolts and plus 116 millivolts, respectively. However, after the fourth injection of dinitrophenol a marked elevation of the potential was maintained, averaging for the aqueous and the lens plus 168 millivolts and plus 159 millivolts, respectively, which persisted after cessation of the treatment.

J. E. LEBENSOHN.

Conjunctiva

THE EYE AND GENERAL INFECTIONS: CONJUNCTIVITIS AND GONOCOCCIC ARTHRITIS. E. PUSCARIU and D. LĂZĂRESCU, *Ann. d'ocul.* 173: 893 (Nov.) 1936.

Infection in the eye is met with in two forms: first, the exogenous form (the more frequent) and, second, the endogenous form, which is responsible for metastatic ophthalmic lesions. The pathogenic agents may penetrate into the organism after entering the eye. Many references are given in the literature to observations on septicemia, meningitis, abscesses or arthritis in which the eye served as the point of entrance to such pathogenic agents as streptococci, staphylococci and pneumococci.

From the statistics of the ophthalmologic clinic at Jassy the following figures are taken: From 1919 to 1935, among 37,960 patients there were found 461 with purulent ophthalmia (1.02 per cent). When the total number of cases of conjunctivitis is considered, the percentage is 4.09 per cent. The patients were examined, as a rule, from the seventh to the thirtieth day after the beginning of the ophthalmia, but generally in the last week. Among the 461 patients with gonococcic conjunctivitis, there were 3 in whom the condition was complicated with arthritis—2 babies and the mother of one of these. Observations on these 3 patients are given in detail. One of the patients had polyarthritis; in the other 2 the localization was mono-articular.

After treatment with antigonococcus vaccine, intramuscular injections of milk in the babies, and infra-red rays, the articular lesions were considered cured. An extensive bibliography is given.

S. H. MCKEE.

KERATITIS PUNCTATA SUPERFICIALIS AND SWIMMING POOL CONJUNCTIVITIS W. P. LING, *Chinese M. J.* 50: 1381 (Oct.) 1936.

This article consists in the report of fifty-three cases of keratitis punctata superficialis in Chinese patients, in nearly half of whom the condition was associated with swimming pool conjunctivitis, which was particularly prevalent in Nanking during the summer and autumn of 1934. There appeared to be a relationship between the keratitis and the swimming pool conjunctivitis, although the former could occur independently of the latter. It is possible that both these conditions had the same etiology, which, of course, is still unknown.

The simultaneous occurrence of these two conditions in the same patient is new to the author and does not seem to have been mentioned in the literature. One must bear in mind that swimming pool conjunctivitis is not confined to swimming pools. As the infection is probably of genito-urinary origin it can naturally occur under any circumstances and may be transmitted from one person to another at any time. Accordingly, some authors have suggested the name "adult inclusion conjunctivitis," or "inclusion blennorrhea of the adult."

S. H. McKEE.

Congenital Anomalies

A CASE OF DISSOCIATED ALBINISM. J. BOLLACK and DELTHIL, Bull. Soc. d'opht. de Paris, October 1935, p. 623.

A woman of 29 years showed on oblique illumination of the eyes a chestnut-brown iris with a red coloration of the pupil. Both the iris and the sclera were especially transparent to reflected light. Horizontal nystagmus was present in all aspects. The entire fundus was pale, as though the retina rested on the detached network of the choroid. In the intervacular spaces of the choroid there was no evidence of pigmentation. There was no differentiation of the macula, and no pigment was present in this area. The fovea was indistinct, and only tortuous vessels could be seen. The normal reflex of the retina was entirely absent, particularly at the fovea. There was no pigment in the posterior layer of the iris, but the stroma had its normal brown pigment, and the entire uvea was translucent. The contour and reaction of the pupil were normal. The cornea, lens and vitreous were normal.

L. L. MAYER.

Cornea and Sclera

CONTACT GLASS AS A THERAPEUTIC AGENT IN CORNEAL ULCERS. D. M. ROLETT, Am. J. Ophth. 19: 888 (Oct.) 1936.

Rolett reports two cases of corneal ulcer, in one of which the ulcer was associated with hypopyon and in the second of which it was due to neuroparalytic keratitis and lagophthalmos. In the first case atropine and strong protein silver were used in the contact glass, and in the second case Ringer's solution was used. Both patients were discharged as cured in ten and nine days, respectively. Rolett calls attention to the simplicity of this treatment and its brilliant results.

W. S. REESE.

TRANSPLANTATION OF CORNEA. J. W. T. THOMAS, Irish J. M. Sc., July 1936, p. 289.

Thomas has done thirty-six consecutive operations for corneal transplantation, twenty-four of which were performed on twenty-one suitable eyes. About 80 per cent of the patients were benefited by the operation; in 28 per cent the grafts were clear, and vision was 6/36 or better. Seventy per cent of the operations were successful, and 25 per cent resulted in clear grafts. The author quotes the results of Elschnig,

Filatov and Castroviejo, from which it is seen that the operation for corneal transplantation is one that should be included in the list of operative procedures which can be legitimately employed in suitable cases. The study of the operation is yet far from completed. Attention to detail is an essential requirement, and minute experimental departures from the routine procedure should be tried singly and not collectively, so that a logical inference may be drawn from the result. The outlook in the future for patients more or less blind from corneal opacities is decidedly encouraging, and in suitable cases there is even now a 70 per cent chance of improvement and a 25 per cent chance of obtaining good useful vision. The future will hold some chance of improvement in many of the cases in which the condition is, for various reasons, classified as unfavorable.

J. A. M. A. (W. ZENTMAYER).

KERATITIS ULCEROSA AND NONULCEROSA PRODUCED BY DIPLOBACILLUS PETIT. P. S. SOUDAKOFF, Chinese M. J. 50: 1415 (Oct.) 1936.

This article consists in the report of three cases of corneal ulcer in which a relatively rare micro-organism was found to be the cause. The diplobacillus of Petit was first described by him in 1898, and was studied serologically by Chaine in 1914, who proved that it was not an evolutionary form of the diplobacillus of Morax and Axenfeld but a variety of the same family.

Keratitis produced by the diplobacillus of Petit either takes a mild course, the patient showing multiple nonulcerating infiltrates of about three weeks' duration, or appears as crescent-shaped relatively benign ulcers. The condition generally reacts satisfactorily to conservative treatment.

S. H. McKEE.

Experimental Pathology

SOME REMARKS ON OEDEMA. J. A. VAN HEUVEN, Brit. J. Ophth. 20: 631 (Nov.) 1936.

After recounting the effects of instilling oleum sinapsis into the conjunctival cul-de-sac van Heuven comments on the chemosis which results. It might be thought that liquids reach the tissues. However, it appears to be impossible to recover liquids by means of puncture, incision or squeezing. The use of vital staining was tried, a 1:800,000 solution of fluorescein and a 1:1,000,000,000 solution of acriflavine hydrochloride being employed. The examinations were made with the biomicroscope under arc light illumination through quartz glass and Uvet filters. The injections were given intravenously after instilling oleum sinapsis. From these experiments it appears that in inflammations the normal circulation of the blood is disturbed, as well as that of the lymph. In the lymph this change is opposite to that of the blood. Without vital staining there is an increase in the efferent current, whereas with vital staining there is an increase in the afferent current. In inflammation there is an increase in the permeability of the vessels, and the liquid leaves the vessels and is incapable of reentering them. It is through the lymph vessels that the liquid leaves the tissues; the

capacity of the lymph vessels is insufficient to absorb the total amount, and the chemical picture of chemosis results. Many other interesting questions in connection with these experiments are discussed.

W. ZENTMAYER.

General

A CRITICAL SUMMARY OF SURGICAL EXPERIENCES IN 1934. E. HILL and R. H. COURTNEY, *Am. J. Ophth.* 19: 773 (Sept.) 1936.

This article does not lend itself to abstracting. It is a report of the experiences of the ophthalmic staff of the Medical College of Virginia during 1934 in both private and clinical practice, based on 239 operations.

W. S. REESE.

OPHTHALMOLOGY AND RESEARCH. JOHN PARSONS, *Brit. J. Ophth.* 21: 1 (Jan.) 1937.

This is an address delivered before the American Academy of Ophthalmology and Otolaryngology. It does not well lend itself to abstracting. The introductory remarks are on vision, and it is pointed out that the study of normal vision demands no small knowledge of physics, chemistry, physiology, neurology and psychology.

The present status of the theory of the function of the intra-ocular fluid is discussed. Much of the evidence to show that osmotic interchange is more important than was once thought depends on minute physical and clinical measurements which are perilously near the borderline of error. Parsons states that he is always rather suspicious of chemical weighing carried to four decimal places!

The newer view concerning the nature of the vitreous is discussed, and the chemistry of the lens and cataract are considered. More recent studies of the circulation of the fluids and the cause of glaucoma are reviewed. Parsons has an open mind on most of these subjects. The paper will well repay reading.

W. ZENTMAYER.

General Diseases

CLINICAL ASPECTS OF OCULAR TUBERCULOSIS. F. MEHLMACK, *Arch. f. Augenh.* 110: 39, 1936.

Three hundred and thirty-five cases of ocular tuberculosis are reported from Hertel's clinic in Leipzig. The largest percentage of patients were between 20 and 50 years of age. The majority of the patients between the twentieth and thirtieth decades were women. The uveal tract was the seat of the disease in 80 per cent, and the patients apparently suffered chiefly in two periods of life, either early in youth or later (in the case of women, at the time of the menopause). In practically all the cases of uveal tuberculosis primary lesions were found in the chest. The tuberculin reaction was positive in practically all the cases in which clinical and roentgen examination revealed inactive tuberculosis, but in a number of the very young patients and in some of the women during the menopause the reaction was negative in spite of definite evidence of active generalized tuberculosis, such as involvement of the lymph glands with or without pulmonary involvement.

Mehlmack does not believe that the exudative form of uveal tuberculosis is limited to any one age group. A general résumé of the treatment as carried out in Hertel's clinic is given. Sixty-five per cent of the patients were cured.

F. H. ADLER.

Glaucoma

CHRONIC GLAUCOMA WITH LOWERED ARTERIAL TENSION, TREATED WITH A CALICUM-MAGNESIUM PREPARATION. JEAN GALLOIS, *Bull. Soc. d'opht. de Paris*, December 1935, p. 801.

A patient of 61 years had bilateral chronic glaucoma with the usual symptoms. Pilocarpine controlled the tension, but the halos persisted, as did an arterial pulsation. Later, visual acuity became reduced, and the question of an operation arose. Resort to injections of a calcium-magnesium preparation as recommended by P. Viard resulted in complete control of the visual acuity, visual fields and tension. A chart showing the tension is included. The drug is used in the following combinations: calcium chloride, 0.75 Gm.; magnesium thiosulfate, 0.8 Gm., and water, to make 10 cc.

L. L. MAYER.

ANTERIOR CHAMBER PUNCTURES: AN AID IN THE DIAGNOSIS OF GLAUCOMA. P. C. KRONFELD and LIN CHING-K'UEI, *Chinese M. J.* 50:1323 (Oct.) 1936.

The writers describe the technic of, and past experiences with, puncture of the anterior chamber in the diagnosis of primary glaucoma in the early stages. The qualifications for Chinese control cases are discussed, and the results obtained in the tests on the controls are given.

In twelve patients with primary glaucoma in the early stages or with signs suggestive of this condition, puncture of the anterior chamber was performed; this resulted in a typical and more pronounced hypertensive reaction than that obtained in the controls, in ten of the twelve cases.

Puncture of the anterior chamber is recommended as a safe and expedient provocative test for the detection of early or latent primary glaucoma. A bibliography is given.

S. H. McKEE.

Hygiene, Sociology, Education and History

OCCUPATIONAL ABILITY OF INDIVIDUALS OPERATED ON FOR CONGENITAL CATARACTS. K. VOM HOFE, *Arch. f. Augenh.* 110:35, 1936.

Of twenty-one patients with bilateral congenital cataracts who had been operated on, ten had good visual acuity. Nine had sufficient visual acuity to pursue their usual occupation. In only two was the visual acuity insufficient for the patients to learn a trade. In the vast majority vom Hofe found the visual acuity sufficient for the following employments: locksmith, carpenter, merchant, farmer, messenger, servant, student, clergyman.

F. H. ADLER.

A SHORT HISTORY OF OLD CHINESE OPHTHALMOLOGY. LEE TAO, Chinese M. J. 50: 1513 (Oct.) 1936.

A brief historical sketch of Chinese ophthalmology is given, and the suggestion is made that a great part of Chinese ophthalmology is based on Indian medicine. The knowledge of the anatomy of the eye was scanty, and diseases of the eye in most cases were ascribed to internal diseases.

Many diseases of the eye are mentioned in ancient Chinese literature, and the different forms of treatment given for most of them may be considered to be more or less useless.

S. H. McKEE.

Injuries

THE MINOR SEQUELAE OF EYE CONTUSIONS. M. DAVIDSON, Am. J. Ophth. 19: 757 (Sept.) 1936.

From a study of thirty-four contusions of the eye and an analysis of the sequelae Davidson found a prevailing concomitance of from two to six lesions, obviously anatomically related to each other and stretching from the root of the iris along the equator of the lens, the zonule, the ciliary body, the ora serrata and the vitreous which justifies the notion of the syndrome of trauma of the anterior segment described by Henri Frenkel. This is of medicolegal importance in the presence of a lesion of the anterior segment. Davidson discusses the different theories of contusion and some of the problems in its mechanism that still require study.

W. S. REESE.

ACCIDENTAL FREEZING OF THE EYE BY SULPHUR DIOXIDE. C. P. CLARK, Am. J. Ophth. 19: 881 (Oct.) 1936.

Clark gives the following summary:

"1. The history and clinical phenomena are related of two eyes that received serious impairment from a sudden jet of liquid sulphur dioxide.

"2. The result of this accident was sudden and intense freezing of the ocular structures.

"3. There was a central facet in each injured cornea.

"4. The following treatment is suggested: Wash the eye at once with cold water (ice water would be preferable) and follow with the application of iced compresses. Local anesthetic and cycloplegic drugs should be employed in sufficient amount to maintain their therapeutic effect. An antiseptic ointment should be applied between the eyelids. Emphasis is given to the prompt application of cold water to the injured eye because if this is delayed much greater tissue damage will result."

W. S. REESE.

IMMEDIATE DAMAGE TO THE IRIS AND LENS FROM AN AMMONIA BURN. W. KISS, Arch. f. Augenh. 110: 98, 1936.

An eye in which a 10 per cent solution of ammonia had been splashed presented the following appearance when first seen in the clinic:

The conjunctiva was gray and opaque, with many hemorrhages scattered throughout. The corneal epithelium had entirely disappeared, and the eye was insensitive. The anterior chamber was deep and gave a Tyndall effect. The iris was semidilated and did not react. At the upper margin the stroma was pushed forward, and the pigment epithelium, which had become separated from the stroma, hung down in the form of a curtain, in folds. The lens showed some indefinite clouding.

Ten months after the injury the eye was enucleated for cosmetic reasons. The layers of the iris were found to be separated by a transudate, which Kiss believes came from the vessels of the iris when the strong solution of ammonia diffused through into the anterior chamber.

F. H. ADLER.

INFLUENCE OF RED LIGHT ON THE EYES OF PHOTOGRAPHY WORKERS.

D. M. NATANSON and D. P. VINOGRADOW, *Sovet. vestnik oftal.* 8: 852, 1936.

Seventeen photography workers were examined during the working hours (from four to eight hours). Anomaly of refraction was observed in fifteen; the color sense was normal in all, and accommodative asthenopia was observed in four. The light sense, which was examined carefully, was found to be reduced to one fourth of normal. The common pathologic change in all the workers was chronic conjunctivitis, which evidently was caused by prolonged work under poor lighting and by the action of red and infra-red rays on the conjunctiva.

O. SITCHEVSKA.

Lacrimal Apparatus

RATIONAL THERAPY OF PATIENTS WITH CATARRHAL CONDITION OF THE LACRIMAL APPARATUS. I. V. SERGEEV, *Sovet. vestnik oftal.* 9: 222, 1936.

Since Sergeev considers stenosis of the lacrimal punctum one of the chief causes of epiphora, he uses the following surgical method for correction of this condition: The lower punctum is dilated with a dilator; a probe, size 1, is introduced up to the nasal bone, the lumen being dilated as much as possible by pulling the lower lid down and out. Then a discission knife is introduced with its cutting surface upward, and the medial wall of the canaliculus is incised for from 2 to 3 mm. The probe and knife are removed; the lateral lip of the wound is grasped with forceps, and a triangular piece is cut out with scissors so that a wide opening of the punctum is made. A fibrinous blood clot forms under the dressing, which is removed in two or three days.

One hundred and twenty-nine patients were treated by this method. Seventy, or 55 per cent, were cured; in fifty-three, or 41 per cent, the epiphora was partially improved, and in four there was no change in the condition. The visits to the dispensary were reduced to one-fourth. The majority of the patients were observed for about eight months.

O. SITCHEVSKA.

Lens

ANTERIOR LENTICONUS. E. C. MOULTON, J. A. M. A. 107: 1809 (Nov. 28) 1936.

According to Moulton, the case here described is the eleventh of the cases of the true type of anterior lenticonus reported. The patient was a man aged 33. Since the age of 17 he had been aware of poor vision. Vision in a darkened room was 20/100 (6/30) in the right eye; with a + 1.00 sphere it was 20/70 (6/21). In the left eye it was 20/70 (6/21); with a +1.00 sphere it was 20/50 (6/15).

With the ophthalmoscope the characteristic "oil drop" was observed in the center of the pupil. With the pupil under the influence of atropine the fundus was plainly seen through the peripheral emmetropic lens substance. Through the apex of the cone the fundus was plainly seen with a -20.00 lens. No changes in the fundus were seen.

Iridectomy was performed in the lower nasal quadrant of the iris of the left eye. The resulting vision was 20/50 (6/15) and was not improved by a glass.

With the biomicroscope two zones of disjunction were seen to be involved, the anterior band of the lens and the anterior band of disjunction. These two bands were seen to bend forward over the clear underlying contacts of the conus, without alteration in their parallelism or change in the thickness of either zone. All the other zones were normal. On the apex of each zone and situated just beneath the intact anterior capsule were a few very minute and delicate brown granular opacities.

In the three cases of this condition reported to date in which the eye was examined with the slit lamp the disorder must have been the acquired form of anterior lenticonus, and in this case, as in the other two, it is a question rather of lentiglobus than of lenticonus.

W. ZENTMAYER.

INTRACAPSULAR CATARACT EXTRACTION. O. M. DUTHIE, Brit. J. Ophth. 21: 16 (Jan.) 1937.

Duthie employs the technic of Sinclair modified only in minor details. Retrobulbar injection of an anesthetic is given twenty minutes before operation. An injection is also made into the lids, and subcutaneous injections are given in order that retraction sutures, which are used in place of a speculum, may be inserted painlessly. A large corneal section with a small conjunctival flap is made. The capsule of the lens is grasped as near the level of the lower pupillary margin as possible. Where the vitreous tends to bulge after extraction of the lens, gentle pressure is applied to the globe, the lids being closed, for one to two minutes. A peripheral iridectomy is preferred.

The author gives the following summary:

"1. The present paper deals with a series of 100 consecutive intracapsular cataract extraction operations.

"2. The results show a final visual acuity, in healthy eyes, of 77 per cent, 6/6 or better, and 19 per cent, 6/9-6/12.

"3. The most common single complication is hyphaemia—28 per cent—for which no suitable method of prevention is suggested.

"4. Iris prolapse occurred in 8 per cent and vitreous prolapse in 6 per cent of cases; suggestions are made with a view to reducing this incidence and at the same time making peripheral iridectomy a safer procedure.

"5. Minor points in local anesthesia are suggested.

"6. Superiority of the operation over other methods is claimed, particularly in immature cases."

W. ZENTMAYER.

HEREDITARY LUXATION OF THE LENS AND THE SYNDROME OF MARFAN.

DELORD and VIALLEFONT, Bull. Soc. d'opht. de Paris, January 1936, p. 44.

A father aged 41 and his son aged 15 years showed various stigmas of the syndrome of Marfan. Both had bilateral luxation of the lens. Scoliosis, arachnodactylia, hyperactive reflexes, absence of secondary sexual characteristics, infantile testicles, a basal metabolism lowered 25 per cent and a blood calcium content of 0.13 per cent were found in the son, but the father was normal in all these respects. Delord and Viallefont discuss the various theories of the etiology of the condition. Excellent photographs of the son are shown in the article.

L. L. MAYER.

BIOMICROSCOPY IN TWO CASES OF DYSTROPHIC MYOTONIC CATARACT.

G. VON GROLMAN, Arch. de oftal. de Buenos Aires 10:707 (Sept.-Oct.) 1935.

This is the report of a biomicroscopic examination of cataract in a sister and a brother of 31 and 34 years with dystrophic myotony. In the sister, who had abundant spherules of shagreen on the anterior capsule and a diffuse opacity of the cortical layers, the light reaching the superficial layers of the adult nucleus, the anterior suture wheel, divided into eight spokes, was quite clear; the diffuse opacities were uniformly distributed in a superimposed layer with bright reflection of light in the central and equatorial regions. The brother showed in the right eye a normal anterior capsule; the cortical opacity was uniform with spots of denser opacity in the form of facets, and there were white spots of a dull aspect in the equatorial zone. The anterior suture of the fetal nucleus was visible, and the fetal nucleus was more opaque than the adult; on the anterior Y bright points of a red, green or blue color according to the incidence of the light could be seen (Vogt's points). In the left eye of the same patient the anterior capsule was normal, with some spherules of shagreen; there was a diffuse dustlike cortical opacity arranged in layers and as a dense pointlike formation; direct illumination showed clearly that the fetal nucleus was much more opaque than the adult, and the anterior suture was clearly visible.

C. E. FINLAY.

THE AMOUNTS OF ACID-SOLUBLE ORGANIC PHOSPHATE IN NORMAL AND CATARACTOUS LENSES. H. K. MÜLLER, Arch. f. Augenh. 109:497, 1936.

Müller has proved that in the lens cevitic acid is formed from sugar according to the following equation—sugar \rightarrow oxidized gluta-

thione = cevitamic acid + reduced glutathione. On the basis of other work he believes that there is a close relationship between vitamin C and the amount of carbohydrate-phosphatide present in the lens, and the present work was undertaken to disclose this relationship.

The phosphatide content of young and old ox lenses was determined by the method of Fiske and Subbarow (*J. Biol. Chem.* 66: 375, 1925), and it was found that young lenses contain more acid-soluble organic phosphate and more vitamin C than old lenses. Similar determinations were made on cataractous lenses from oxen, and it was found that less acid-soluble organic phosphate was present than in the normal lens.

Müller concludes that in the development of a cataract the lens not only loses vitamin C but also its acid-soluble organic phosphorus. On the other hand, the inorganic phosphate is usually not reduced in amount, and frequently more is found than in the normal lens, which corresponds well with the increase in calcium known to occur in cataractous lenses.

F. H. ADLER.

Lids

REMOVAL OF THE TARSAL PART OF THE ORBICULARIS MUSCLE OF THE UPPER LID FOR TRACHOMATOUS ENTROPION AND TRICHIASIS. A. BUSACCA, *Ztschr. f. Augenh.* 88: 100 (Jan.) 1936.

Busacca believes that the entropion and trichiasis complicating trachoma are due chiefly to hypertrophy of the marginal part of the orbicularis muscle, the hypertrophy coming from the spasm of the lid caused by the primary keratitis.

The removal of the muscle bundle is done through a single incision in the skin 0.5 cm. from the margin of the lid. The tarsus is thinned down to leave a step at the upper margin where the levator attaches. The lower edge of the skin is stitched to this step with five or six sutures. The upper edge of the skin is left free to form the normal fold of the lid.

The operation accomplishes the removal of hypertrophic muscle and holds the margin of the lid and the lashes away from the cornea. Several hundred patients have been treated by this method in the last four years, and no cases of recurrence have been observed by Busacca. Explanatory drawings accompany the article.

H. GIFFORD JR.

Methods of Examination

THE CLINICAL VALUE OF TRYPTOPHANE-REACTION. J. A. VAN HEUVEN, *Brit. J. Ophth.* 20: 635 (Nov.) 1936.

As a result of experimental study van Heuven concludes that the tryptophane reaction has been proved to be not a specific test of tuberculosis but a sensitive indication of increased permeability of the capillaries. An advantage of the reaction is that it allows one to demonstrate increased permeability by means of a substance normally existing in the circulation.

W. ZENTMAYER.

EXAMINATION OF THE EYE BY MEANS OF THE SODIUM LAMP. G. KLEEFELD, Bull. Soc. d'opht. de Paris, December 1935, p. 788.

A review of the history of the use of the sodium lamp, with some observations on its clinical application, is given. Kleefeld concludes that the sodium lamp presents a certain interest for the ophthalmologist. Its use in the study of the palpebral vessels and the conjunctiva and in focal illumination is of value. The lacrimal papilla is viewed with great ease, and atresias are brought out. Opacities of the lens and vitreous are more readily studied than with ordinary illumination. Because of the suppression of the red color, the fundus is more precisely studied. Localization of hemorrhages, especially preretinal hemorrhages, is easily made. Skiascopy is rendered more exact, and more remarkable contrast is noted in photography of the anterior segment of the eye than with other sources of illumination.

L. L. MAYER.

Neurology

RELATION BETWEEN DISEASE OF THE TRIGEMINAL NERVE AND INTRA-OCULAR OPERATIONS: REPORT OF A CASE. W. GILBERT, Klin. Monatsbl. f. Augenh. 97: 70 (July) 1936.

Gilbert discusses the relations between disturbances in the area of the trigeminal nerve and intra-ocular diseases, with special reference to glaucoma and the divers types of ocular herpes, and also neuralgia. He reports the case of a woman aged 57 to show the serious effect of neuralgia of the first branch of the trigeminal nerve, which developed during recovery after extraction of a senile cataract. The patient had no ocular or other diseases at any time except neuralgia in the right side of the head for many years. Purulent iritis set in three days after the operation, while the corneal flap remained intact. Operation for secondary cataract was performed with De Wecker's scissors seven months later. A hemorrhage in the anterior chamber was observed three days later, and a number of hemorrhages from the temporal pillar of the coloboma of the iris occurred two or three times a week for about four months. These hemorrhages resembled those described by Gilbert in his paper on herpes. Secondary glaucoma, associated with severe attacks of neuralgia, necessitated enucleation. Histologic examination revealed necrosis of the upper portion of the iris and perineuritic inflammation of some ciliary nerves; the choroid and retina were intact. Gilbert thinks that herpes virus entered the trigeminal nerve endogenously, causing chronic inflammation in its branches up to the ciliary nerves. This process caused the neuralgia without involvement of the eye. The operation produced chronic neuritis in the ciliary nerves. Gilbert concludes that extraction of cataract should not be performed prior to six months after the last attack of neuralgia. It is advisable to perform preliminary iridectomy or avoid iridectomy altogether.

K. L. STOLL.

Ocular Muscles

PARALYSIS OF DIVERGENCE OF FUNCTIONAL ORIGIN. C. P. CLARK, Am. J. Ophth. 19:789 (Sept.) 1936.

Clark reports the case of a 26 year old white woman who under stress of economic and family troubles began to suffer from aching of both eyes and then diplopia. There was concomitant convergent strabismus, the deviation at 6 meters varying from 12 to 25 degrees. Fixation alternated between the two eyes, and the field of rotation for each eye was normal when the fellow eye was occluded. Binocular single vision was present when the test object was brought to within 60 or 70 cm. of the face. The near point of convergence was 6 cm. The condition eventually cleared up almost entirely. The Wassermann test and physical examinations gave negative results.

W. S. REESE.

A CASE OF CONVERGENCE SPASM. P. BAILLIART, Bull. Soc. d'ophth. de Paris, February 1936, p. 81.

A girl aged 8 years had complained of double vision for eight days. The mother reported that the child had had such an attack twice previously, with accompanying convergent strabismus and apparent diplopia. The first attack was at the age of 2 years and lasted eight days; the second, which occurred after a year, was of short duration. These two attacks were thought to be due to eating chocolate. In the present attack the strabismus was 40 degrees. A lens to correct a refractive error of 1 diopter brought the visual acuity of the right eye to 10/10 and that of the left to 9/10. When the child put on her glasses (a + 1.0 diopter sphere) the convergence disappeared. Also, if her attention was distracted or she became greatly interested in something, no evidence of crossing was noted. Bailliart injected a 2 per cent solution of procaine hydrochloride into the right internal rectus muscle; this caused complete disappearance of the squint. Other facts of the history were unimportant. Physical examination gave entirely negative results. This included a complete neurologic examination. Both parents were examined. Bailliart thinks that the condition was spasm of convergence, which has been rarely recorded in the literature, as shown by the reports. In some cases the condition was noted after encephalitis. No etiologic factor was found in the described case.

L. L. MAYER.

Operations

CYCLECTOMY. L. VON BLASKOVICS, Ztschr. f. Augenh. 88:75 (Jan.) 1936.

A rather simple method for removal of a piece of ciliary body through a meridional incision is described. The horse hair sutures are placed through the edge of the sclera before it is cut completely through. After completion of the scleral incision the ciliary body is partly

detached with a cyclodialysis spatula, pulled into the wound and cut off with scissors. The sutures are carried into the conjunctiva so that its closure does not fall opposite the scleral wound.

Five cases are reported in which part of the ciliary body was removed. No serious complications developed that could be attributed to the operation. In one patient 23 years of age who had sustained a trauma of the eye in whom good vision was retained, there was no receding of the near point, and accommodation was undisturbed following cyclectomy.

Blaskovics feels that this is the operation to be chosen in removing nonmagnetic foreign bodies and in cases of rupture of the sclera with prolapse of uveal tissue. If there is no uveal tissue left incarcerated in the wound, he feels that there is no more danger of sympathetic ophthalmia occurring after this operation than following cyclodialysis.

H. GIFFORD JR.

Orbit, Eyeball and Accessory Sinuses

SYNDROME OF NASAL NERVE FROM BACILLARY TOXEMIA. C. CHARLIN, *Ann. d'ocul.* 173: 25 (Jan.) 1936.

Charlin states that he knows well the tuberculous toxemia with loss of weight, asthenia, loss of color and sometimes vesper fever which occurs without clinically observable pulmonary lesions. Fluoroscopy and better still roentgenography show thoracic pulmonary shadows, especially at the hilus. The condition is generally found among young adults, especially females. In many cases with rest and tuberculin therapy he has obtained remarkable cures. He has often been consulted for blepharitis, for blepharokeratitis and particularly by adults for iritis and cyclitis. He observed a syndrome of the nasal nerve in two patients who both to his great surprise gave positive reactions to the Mantoux test. He recollects that nasal neuritis gives a syndrome characterized by (a) symptoms in the anterior pole of the eye (ulcerative keratitis, iritis), (b) symptoms of acute rhinitis and (c) ocular neuralgia, orbital, nasal or frontal. After giving details of the two cases mentioned, Charlin concludes that bacillary infection of nasal origin should be considered in all ocular disorders of obscure etiology and that this infection may exist without apparent signs, particularly in adults and particularly in females approaching the menopause.

S. H. MCKEE.

PRIMARY ACUTE OSTEOMYELITIS OF THE ORBIT. R. ONFRAY and F. MUNCH, *Bull. Soc. d'opht. de Paris*, April 1936, p. 311.

Primary acute osteomyelitis of the orbit is a rare condition which was described by Mackenzie and Lammelongue, and also by Rollet in the "Encyclopédie d'ophtalmologie." The case reported was particularly dramatic and severe and shows the importance of a precise diagnosis and early treatment.

A youth of 18 years was seized with sudden severe pain in the right orbit, extending around the orbital rim. This followed a light

cold. There was no especial discomfort in the nose. The next day the temperature rose to 40 C. (104 F.); the lids were red and swollen, and acetylsalicylic acid did not relieve the pain or the fever. After another twenty-four hours marked chemosis of the conjunctiva occurred; the eye was proptosed and fixed in position. The media of the eye were clear, but the disk was congested, and the veins were slightly tortuous. Examination of the sinuses gave negative results, as did that of the teeth. There was no evidence of thrombophlebitis. Pus suddenly pointed at the superior-internal angle of the orbit, where an incision was made. The subsequent course was stormy. Culture showed predominance of *Staphylococcus*. General complications were pleurisy and endocarditis. It was finally necessary to operate and secure complete drainage from under the periosteum.

L. L. MAYER.

TUBERCULOUS PANOPHTHALMITIS. A. B. KATZNELSON, *Sovet. vestnik oftal.* 9: 205, 1936.

Tuberculous panophthalmitis is differentiated from other tuberculous infections of the eye by the predominance of exudative and purulent inflammation over the proliferation of specific tissue and by caseation and necrosis of tissue, with fistula formation. An active pulmonary process is present simultaneously.

This is the eighth case of this condition reported in the literature. [In a footnote Katznelson cites the case of a ninth patient, who died from meningitis after enucleation of the eye.—Abstractor.]

An 18 year old youth suffered from chronic pulmonary fibrous tuberculosis with an acute exacerbation—"pneumonia"—which was followed by loss of vision in the left eye during a period of two months.

Examination revealed moderate exophthalmos; in the lower temporal quadrant a yellowish round tumor was seen through the conjunctiva. The pupil was covered with a thin exudate; the iris was atrophic; tension was plus. A week later the granuloma softened, and on incision of the conjunctiva a cavity filled with necrotic tissue presented itself; it was connected with the vitreous. A guinea-pig given an injection of pus showed miliary tuberculosis at autopsy. A culture on egg medium yielded tubercle bacilli. A fistula formed at the site of the incision, and phthisis bulbi developed. The general condition and the process in the lungs improved after the fistula formed. The patient was discharged and began working.

Katznelson believes that all the toxic signs—the elevation of temperature, the loss of weight and the arthritis—were due to the "leading" active process in the eye in the form of subacute panophthalmitis with cavitation of the eyeball. The primary metastasis was in the choroid, where a solitary tubercle formed, which later showed conglomeration along the uveal tract. Detachment and necrosis of the retina followed; then necrosis of the vitreous and, finally, necrosis of the sclera with fistula formation occurred, so that true open tuberculosis of the eyeball was present in this case.

O. SITCHEVSKA.

Physiologic Optics

A NEW THEORY OF ANASTIGMATIC SPECTACLE LENSES. F. OSTWALT, *Arch. f. Ophth.* 135: 610 (July) 1936.

In several papers (*Bull. et mém. Soc. franç. d'opht.* 47: 36, 1934; *Ztschr. f. ophth. Optik* 22: 166, 1934; 23: 132, 1935) Ostwalt modified his original theory of point-focal lenses. These modifications were criticized by Sundgvist (*Arch. f. Ophth.* 135: 67, 1936). Ostwalt now replies and very convincingly states again the reasons why point-focal lenses should be constructed in such a way that pencils of rays which aim at the image of the center of rotation of the eye produced by the combination of the spectacle lens with the eye are free from the astigmatism of oblique incidence.

P. C. KRONFELD.

Physiology

ON THE NATURE OF THE OCULAR FLUIDS. K. MEYER and J. W. PALMER, *Am. J. Ophth.* 19: 859 (Oct.) 1936.

Meyer and Palmer discuss the origin of the intra-ocular fluids, and report some experimental data. They give the following summary:

"Evidence for the dialysis theory of intraocular-fluid formation has been discussed. A polyuronic acid from the vitreous humor and its physico-chemical characteristics have been described. Highly viscous preparations of the acid have been obtained; its viscosity largely accounts for that of the vitreous humor. Evidence for the existence of this acid in the aqueous humor has been presented, which indicates that the intraocular fluid is at least partially formed by a specific secretion."

W. S. REESE.

Retina and Optic Nerve

SPASMS OF THE CENTRAL ARTERY OF THE RETINA. JEAN SÉDAN and G. E. JAYLE, *Ann. d'ocul.* 173: 865 (Nov.) 1936.

The authors continue their article from the August 1936 issue of *Annales d'oculistique* and take up first the question of blindness. They discuss first binocular and then monocular blindness, illustrating the discussion with the histories of patients seen by them. From this they draw the following conclusions: Recurrent retinal spasm, whatever the cause, is always a transitory symptom of a local or general disturbance. It never has the tendency, even in the most intense forms, to provoke definite trouble.

The second part takes up mixed forms of visual troubles of spasmodic origin. These are subdivided into recurrent visual disturbances subsequently complicated by definite lesions, and prolonged and recurrent visual disturbances. Numerous examples are given in each subdivision. The article is to be continued.

S. H. McKEE.

DETACHMENT OF THE RETINA: AN ARRANGEMENT FOR TRANSLUMINATION OF THE SCLERA AND DIATHERMY SIMULTANEOUSLY; AN EXPERIMENTAL CASE. J. LIJO PAVIA and M. DUSSELDORP, Bull. Soc. d'opht. de Paris, October 1935, p. 649.

The scleral lamp of Lange was equipped with diathermy electrodes and used in experiments on animals to show the method of localizing a hole and treating it at once with the current. Pictures of the instrument and of fundi of animals which had been operated on are included in the article.

L. L. MAYER.

METHODS IN THE ACTUAL TREATMENT OF DETACHMENT OF THE RETINA. P. BAILLIART, Bull. Soc. d'opht. de Paris, July 1935, p. 507.

Bailliar reviews the history of the treatment of detached retina. He believes that advance in the methods of treatment is shown by Weve's statistics on cases in which reattachments were obtained, viz.:

1930-1931.....	48%	1933.....	72%
1932.....	63%	1934.....	75%

He concludes that, as a rule, eyes with detachment of the retina should be operated on.

L. L. MAYER.

MEDICOLEGAL CONSIDERATION OF TRAUMATIC DETACHMENT OF THE RETINA. D. SABBADINI, Bull. Soc. d'opht. de Paris, July 1935, p. 520.

Professor Sabbadini, as the authority on legal medicine in Italy, presents the important facts on this subject as related to Italian law.

Gonin's review of the clinical varieties of traumatic detachment gives the principal theme for Sabbadini's discussion. The following have medicolegal value:

1. A written statement obtained at the earliest opportunity.
2. An early visit to a competent ophthalmologist.
3. A well identified type of traumatic reaction.
4. Evidence of trauma and its reaction.
5. Exclusion of slight trauma as a cause of detachment.
6. The possibility of fixing the time when a detachment is or is not definitely stabilized.

L. L. MAYER.

TRAUMATIC DETACHMENT OF THE RETINA. L. GENET, Bull. Soc. d'opht. de Paris, July 1935, p. 543.

Genet reports the case of a 16 year old patient to show the difficulty of deciding on the pathologic character of a traumatic detachment of the retina. At the time of injury the fundus could not be seen, owing to disturbance and clouding of the vitreous. Twelve days later the media were clear, but no retinal change was noted. Twenty days following the injury a detachment of the retina, with disinsertion superiorly, was noted.

Another patient, who was seen in consultation with Dr. Parifque, had a piece of metal strike the eye. He was taken to the hospital at once; because of the intense edema of the lids, little of the globe was seen. Four days later herniation of the iris and luxation of the lens (which was cataractous in part) into the anterior chamber were noted. Following iridectomy the lens absorbed, and when the fundus could be seen, four months following the injury, the entire lower portion of the retina was detached. Genet stresses the point that after injury of the globe hypotension or hypertension may ensue, followed or accompanied by detachment of the retina. In all operations for retinal detachment he has found that the subretinal fluid had been under tension. Iritic reaction following injury to the globe must make the prognosis more guarded; often a detachment of the retina is produced in these cases.

L. L. MAYER.

ETIOLOGY OF THE MACULA. H. HOEHNE, *Ztschr. f. Augenh.* 88: 297 (March) 1936.

Hoehne describes the cases of three patients with macular coloboma who showed definite evidence that this was caused by an inflammatory process, probably tuberculous. He thinks that these cases should be separated from the group of cases of hereditary macular degeneration in which there is always a family history of similar disturbance and from cases of congenital faulty development of the macula in which there is usually evidence of other faulty development, especially of the eyes. The chief causes of the inflammatory type of macular coloboma he lists as acquired and congenital syphilis and tuberculosis.

H. GIFFORD JR.

CHORIO-RETINITIS SCLOPETARIA. T. H. LUO, *Chinese M. J.* 50: 1405 (Oct.) 1936.

The changes in the eyeground which develop following gunshot injuries of the orbit are called chorioretinitis sclopetaria.

As the extensive free bleeding in the vitreous clears up, the following interesting and characteristic picture is revealed: The changes are essentially hemorrhages and whitish patches which involve wide areas in different parts of the fundus. The hemorrhages are preretinal, retinal, behind the retina or in the choroid and are mingled with the white patches in their neighborhood. The white patches are large and irregular but have circumscribed margins. They send out long processes in different directions in the fundus and are often interrupted by small round or oval sharply outlined light red areas.

The article has a colored drawing illustrating the condition.

S. H. McKEE.

Trachoma

TRACHOMA. S. S. MEIGHAN and MARY URQUHART, *Brit. J. Ophth.* 20: 201 (April) 1936.

This is an investigation into the question of the presence of *Bacterium granulosis* (Noguchi) in cases of trachoma in Glasgow. The

laboratory studies followed the general lines of those of Finnoff and Thygeson. Meighan believes that some of the discrepancies in the results obtained by various workers are due to the fact that some of these workers did not allow sufficient time for growth to progress to the extent of producing visible "colonies" before announcing that the micro-organism in question was not present in the material examined. Again, some of these investigators may not have been conversant with the fact that cocaine inhibits the growth of *Bact. granulosis*.

Meighan's studies showed that this organism was present in a proportion of the cases examined.

W. ZENTMAYER.

TREATMENT OF TRACHOMA IN CHILDREN. WIBAUT, *Rev. internat. du trachome* 13: 174 (Oct.) 1936.

The prognosis for the cure of trachoma in children is much better than that for the cure of trachoma in adults. The requirements are early detection and prolonged and prudent treatment. Children under 5 years of age may show only suggestive thickening of the conjunctival epithelium, but between the ages of 5 and 10 years follicles are generally well developed. These follicles should be attacked by expression, and frottage with a 1 per cent solution of silver nitrate or some other suitable medicament should be continued for a few months thereafter. Strong caustics are to be avoided, and operation is seldom necessary.

J. E. LEBENSOHN.

THE LEUKOCYTIC FORMULA IN TRACHOMA. A. C. MIHAIL, *Rev. internat. du trachome* 13: 193 (Oct.) 1936.

Examination of the blood of those affected with trachoma reveals a slight tendency to an increase of the mononuclear elements—lymphocytes and monocytes, especially the latter. Eosinophilia does not occur except with concomitant parasitic infection.

J. E. LEBENSOHN.

Tumors

EPITHELIAL CYST OF THE IRIS. J. CHARAMIS and SFALAGAKO, *Arch. d'opht.* 52: 167 (March) 1935.

The case reported was that of an epithelial cyst with an actual cystic cavity the structure of which was the same throughout. There was no basal membrane, and the cyst was lined with polygonal and cylindric epithelial cells, each having a central nucleus. On the outside the epithelium was flattened and contained fusiform nuclei. Some iris tissue was adherent to it.

In the majority of cases epithelial cyst of the iris is traumatic in origin, developing at the site of a perforating wound of the cornea or, very often, after operation on the anterior segment of the eye. However, the traumatic cyst as a rule arises from corneal or conjunctival epithelium. There are other cases in which the cyst is nontraumatic and endothelial in origin. In still others it may be congenital, arising

from an ectodermic inclusion. Charamis and Sfalagako call attention to the value of biomicroscopic examination in cases of epithelial cyst of the iris but state that in the final analysis the diagnosis depends on microscopic examination after extirpation.

S. B. MARLOW.

BIZARRE GRANULOMA OF THE ORBIT. E. REDSLOB, Bull. Soc. d'opht. de Paris, April 1935, p. 257.

A 21 year old man had tearing and swelling of the lids of the left eye for three months. Vision was normal in both eyes. A slight chemosis of the bulbar conjunctiva and edema of the inferior lid of the left eye were noted. On palpation one could feel under the lower lid a firm mass, round but not painful. A horizontal incision along the border of the inferior lid revealed a round fatty mass. When the mass was elevated there appeared a well delimited reddish mass fused to the nasal bone. This was easily excised. There was no diplopia following the operation. Grossly the tumor formed a compact mass consisting of confluent dark and clear areas. Microscopically the hue of these areas seemed to depend on the color of the nuclei of the cells. The dark areas were formed by a mass of round cells having much chromatin in their nuclei. Lymphocytes and plasma cells were noted in the dark areas while epithelioid cells and indefinable material were found in the clear spaces. Because the dark areas surrounded the clear spaces the formation was that of a nodule. The muscle fibers were separated by the infiltration and in addition to being discolored were dissociated. Redslob believes the tumor to have been an inflammatory lesion and not a neoplasm. General examination disclosed nothing except an increase of the eosinophils of the blood to 7 per cent. Photomicrographs and a bibliography are included.

L. L. MAYER.

A CASE OF NEURINOMA RETINAE. C. F. M. PIECK, Arch. f. Ophth. 135: 451 (July) 1936.

In an eye removed from a woman aged 75 with the diagnosis of retinal detachment and absolute glaucoma, Pieck discovered a primary malignant tumor of the retina which differed in many respects from those described in the literature. The tumor consisted of several types of cells, which seemed to be derived from ganglion cells, glia cells and nerve cells.

P. C. KRONFELD.

Uvea

ETIOLOGY OF HYALINE VERRUCOSITIES. J. BERNACKA-BIESIEKIERSKA and A. WIECZORCK, Bull. Soc. d'opht. de Paris, October 1935, p. 626.

The authors observed a family of seven children in which two girls and a boy had peripapillar hyaline verrucosities (drusen). Congenital syphilis was in the family, and only in those members having the alterations of the pigment of the retina and choroid characteristic of that disease were the hyaline deposits in evidence. Parsons and Morton considered such deposits as the result of a past inflammatory

process. Iwanoff felt that they were portions of the lamina vitrea of the choroid. Lauber explained them as portions or cells of the pigment epithelium of the retina detached during fetal life. Others maintained that the hyaline verrucosities were due to irregularities in the evolution of the ectoderm and mesoderm. Thus every factor having a bearing on the production of the eye has been given as causative. Three photographs are shown.

L. L. MAYER.

Vitreous

HYALOID CANAL WITH AREA MARTEGIANI AND SYMMETRICAL HYALOID FORAMEN. A. VOGT, *Ztschr. f. Augenh.* 88: 1 (Dec.) 1935.

A case of retinal detachment is presented, with excellent illustrations. An oval opening was seen to be sharply delimited, lying almost over the optic disk. The edge of the opening lay from 6 to 8 diopters in front of the retina. Through the opening the retinal vessels were seen as clearly as through the retinal tear. Running out from the opening were sets of radial folds, which slightly obscured the retinal vessels. The folds were thought to be due to disturbance in the posterior hyaloid limiting membrane. The other eye, in which there was no retinal separation, showed a similar distinct oval opening in the vitreous. These openings were thought by Vogt to represent the posterior hyaloid foramen and prove the presence of the funnel-shaped area martegiani.

This case and experiments with berlin blue on the eyes of animals and young children by Gasser should prove conclusively the presence of the posterior hyaloid canal and foramen and would give an anatomic basis for ring-shaped separation of the posterior limiting membrane of the vitreous.

H. GIFFORD JR.

POSTERIOR RING-SHAPED SEPARATION OF THE VITREOUS. E. KRAUPA, *Ztschr. f. Augenh.* 88: 224 (Feb.) 1936.

Kraupa describes and illustrates two cases of circumscribed holes in the posterior limiting layer of the vitreous as seen with the arc light ophthalmoscope. One was smaller than the optic disk and was placed eccentrically. The other patient showed a pretzel-shaped hole. Kraupa feels that, because of their shape and position, they have no relation to the area martegiani but represent destruction of the posterior limiting membrane of the vitreous, being merely one phase of senile ocular degeneration. He claims priority for describing this condition in 1914 and feels that these changes may either precede or be the result of retinal separation.

H. GIFFORD JR.

Therapeutics

DISEASES OF THE EYE TREATED WITH DIATHERMY. P. M. BATRACHENKO and T. M. ASKALANOVA, *Sovet. vestnik oftal.* 9: 149, 1936.

Nine tables illustrate the results of the treatment in various diseases of the eye. In forty-five patients suffering from atrophy of the optic nerve of varied etiology, only those in whom some nerve fibers

were left intact were partial regeneration of these tissues and improvement of vision and of the visual fields obtained. In atrophy of long standing there was no response to the treatment. In fourteen cases of optic neuritis due to toxic doses of plasmochin and in eleven cases of malarial neuritis diathermy was definitely favorable, as the edema of the disk and retina disappeared and vision improved with this treatment. Diathermy is contraindicated in glaucoma because the intra-ocular tension is raised during the treatment; in four patients an attack of acute glaucoma was caused by the treatment. The albumin content of the aqueous of two patients with glaucoma was examined before and after the treatment; it was increased from 3.26 to 16.5 per cent.

Diathermy was of no value in choroiditis and retinitis pigmentosa. In eight cases of vitreous opacities the treatment gave definite improvement. Diathermy was definitely favorable in two of three cases of sympathetic ophthalmia: The intra-ocular tension was raised, and synechiae were torn loose.

Diathermy is contraindicated in trachoma, as it causes an increase of secretion, hyperemia and the appearance of new blood vessels.

In fifty-nine patients suffering from malarial, arthritic and gonorrheal iritis and iridocyclitis, diathermy acted almost as a specific treatment, as the pain subsided after four or five treatments and formation of synechiae was prevented. Tuberculous and syphilitic iritis did not respond well to the treatment. In eleven cases of traumatic iritis and iridocyclitis, diathermy caused exacerbation of the inflammatory process, with increase of the number of precipitates.

Eighteen patients with incipient cataract responded favorably to diathermy treatment: the vision was improved and remained so during one year. Further study and observation are being conducted.

In scleritis and episcleritis diathermy was definitely indicated, as the duration of the disease was shortened and the bluish nodules disappeared with this treatment. This method of treatment was also successful in cases in which grafts after Thiersh were made and in cases of plastic operations on the lids.

In keratitis (forty-seven cases) of malarial origin, parenchymatous, neuroparalytic and tuberculous, the results were not favorable.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Clinical Meeting, Dec. 11, 1936

MR. W. H. McMULLEN, O.B.E., F.R.C.S., *President*

VASCULAR LESIONS FOUND IN ASSOCIATION WITH MYOPIA: A REPORT OF SIX CASES. MISS J. M. DOLLAR.

CASE 1.—A woman aged 60 was first seen six years ago because of extensive nebulae in the right cornea and myopic changes in the left fundus. A recent examination disclosed visual acuity of 6/24 in the left eye with a minus cylinder, axis 180. In the region of the left macula there was a black circular area, which seemed to be an example of Fuchs' black spot, in spite of the low myopia. The coagulation time was three minutes.

CASE 2.—Retinal hemorrhage associated with high myopia was present in a woman aged 38. When she was first examined, in November 1917, corneal nebulae were seen, due to old interstitial keratitis. In January 1926, there was a hemorrhage in the macular region of the right eye. Eight years later vision with glasses was 6/12 in the right eye and 6/18 in the left. Two years later, in September 1936, she complained that vision of the left eye had deteriorated; with the aid of her glasses she could count fingers at the distance of 1 foot (30.5 cm.). The physician reported that the condition was mild pituitary dystrophy. The Wassermann reaction was negative; the coagulation time was two and one-half minutes. There was a whitish excavated area in the right fundus, at the site of the first hemorrhage.

CASE 3.—A woman aged 44 complained of failing vision in the left eye. In each fundus there were myopic changes. Vision of the right eye was 6/12 and that of the left eye was 6/60 with a minus 9 sphere for each. There was a coagulation time of eight and one-half minutes. The point of interest in this case was an absorbing choroidal hemorrhage in the left eye downward and outward from the disk, in association with high myopia.

CASE 4.—A woman aged 60 complained of poor vision. Choroidal hemorrhages were seen downward and outward from the disk in the left eye, associated with a high degree of myopia. In each fundus myopic changes could be seen. The blood pressure was 120 systolic and 80 diastolic.

CASE 5.—A man aged 49, who was first seen four months ago, complained that vision of the left eye was getting worse. Vision of the right eye with glasses was 6/6; that of the left eye was 6/36. There were retinal hemorrhages and exudates in both fundi. The blood urea

content was 48 mg. per hundred cubic centimeters, but there were no renal casts. The systolic blood pressure was 260; the Wassermann reaction was negative. In this case there were advanced renal changes combined with an absence of definite evidence of renal involvement, as is usual in cases of essential hypertension.

CASE 6.—The case of a woman aged 66 with retinitis circinata was reported. When seen a month ago she said that the sight of the left eye had been getting worse for years. With glasses she could only count fingers with the left eye. No thickening of the radial arteries was evident; the blood pressure was 120 systolic and 60 diastolic. This patient showed the typical lesion.

MACULAR CHOROIDAL HEMORRHAGE: MR. R. M. TRYELL.

CASE 1.—A married woman aged 46 had loss of central vision in the left eye for six weeks. A large mound of one and one-half disk diameters was seen lying above the macula, with a retinal vessel crossing it. The center of the mound has since caved in, leaving an area with a crater-like appearance. Vision improved from 6/36 (at the first attendance) to 6/12.

CASE 2.—Probable macular choroiditis was shown by a woman aged 37. She complained of a central patch of blindness in the left eye, but vision around it was possible. Examination showed the exudative type of choroiditis at the macula but little disturbance elsewhere. In front of the macula was a good deal of exudate. The Wassermann reaction was negative. Several diseased teeth were extracted.

CASES EXEMPLIFYING VARIOUS TYPES OF RETINITIS PIGMENTOSA. MR. JOSEPH MINTON.

CASE 1.—A woman aged 31 complained of night blindness. Vision of each eye was 6/12, but the field of each eye was concentrically constricted to 10 degrees. The disks were yellowish; the retinal arteries were narrowed, and in the periphery a few specks of pigment were visible. The patient's general health was good; there was no history of consanguinity in the parents, and no other members of the family showed ocular abnormalities.

CASE 2.—A woman aged 28 complained of night blindness, which she had noted for three years. Both eyes showed full vision, but the right and the left field were concentrically constricted to 15 degrees. The retinal arteries were narrowed, and one could see the choroidal circulation. Deposits of pigment were scattered in the periphery. The quantity of pigment had been increasing. The general health was good, and there was no history of consanguinity.

CASE 3.—A girl aged 8 years complained of night blindness. Vision in both the right and the left eye was 6/9; the right and the left field were concentrically constricted to 10 degrees. The retinal arteries were much narrowed, and the choroidal circulation was visible. In each fundus fine granules of pigment and large pigmented areas were seen. In February 1935 the patient showed white shining bodies in

the periphery of the fundi and a much smaller amount of pigment. The Wassermann reaction was negative, and the general health was good. No abnormalities were found in the remainder of the family.

OXYCEPHALY IN A PATIENT SHOWING APERT'S SYNDROME. MR. S. R. GERSTMAN.

A boy aged 7 years had a broad, square skull, bilateral ptosis, ankyloblepharon and syndactylia, the second and third, and the fourth and fifth toes being webbed. There was no consanguinity of the parents, but the familial relationship of the syndrome was shown by the syndactylia of the second and third toes of both feet of a female cousin of the patient's father. Vision was good; there was no atrophy of the optic nerve, and the boy was unusually bright.

OXYCEPHALY IN A PATIENT SHOWING CROUZON'S SYNDROME. MR. S. R. GERSTMAN.

A boy aged 12 had oxycephaly and prognathism, a high-arched palate, exophthalmos, coloboma of the irides and atrophy of the optic nerves. Vision without glasses was 6/60 in each eye; vision with glasses was 6/36 in the right eye and 6/18 in the left. The patient was somewhat dull for his age. The maxillae were ill developed.

FAMILIAL PERIPAPILLARY CHOROIDAL SCLEROSIS IN A PATIENT WITH AN EXTENSIVE CONDITION INVOLVING THE CENTRAL AREAS (LAURENCE-MOON-BIEDL SYNDROME). MR. H. AVERY and MR. ARNOLD SORSBY.

A boy aged 16, the fifth son of parents who were first cousins, showed the complete Laurence-Moon-Biedl syndrome, i. e., obesity, hypogenitalism, polydactylism, pigmentary degeneration of the fundi, mental deficiency and a recessive character of the disorder. There were five children of the marriage, all sons. The second son, who had been affected in the same way, died of pneumonia last year at the age of 23.

CORNEAL DYSTROPHY. MR. E. J. SOMERSET.

A boy aged 8 years, since the age of 16 months has had twenty-three attacks of spontaneous erosion of the cornea, characterized by superficial loss of corneal epithelium over an area 6 or 7 mm. in diameter. At the time of erosion there was little injection, and in a few weeks the area had become healed over. Examination showed a very few dotlike opacities in Bowman's membrane.

STARGARDT'S DISEASE. MR. L. H. SAVIN.

A girl aged 15 showed typical pigmentary macular degeneration, without other abnormal signs. The other members of the family were healthy, and there was no consanguinity. In 1932 vision was 6/12; central vision is now lost.

FAMILIAL DISTICHIASIS. MR. L. H. SAVIN.

The cases of two patients with familial distichiasis, aged 3 and 2 years, respectively, were reported. The first patient was greatly troubled by pain and photophobia; therefore the posterior rows of lashes were excised and the raw areas on each lid were filled by grafts of mucous membrane from the lip. The photophobia is now relieved. Suggestions were sought for treating the patient's sister, whose lashes are not yet causing trouble.

DISCUSSION

MR. CHARLES GOULDEN: I should advocate electrolysis, half a dozen lashes being treated at a time.

RUPTURE OF DESCMET'S MEMBRANE OF THE LEFT CORNEA SUSTAINED AT BIRTH. MR. L. H. SAVIN.

A boy aged $2\frac{1}{2}$ years, who was delivered by instruments, showed a scar much less marked than formerly, especially in the posterior layers of the substantia propria of the left cornea. A less obvious linear indentation of the left frontal bone was also present.

REVOLVER SHOT THROUGH BOTH ORBITS. MR. L. H. SAVIN.

A married woman aged 28 accidentally discharged a revolver into the right temporal region, and the bullet emerged through the left eyebrow. When first seen the patient was conscious but could not see. Both eyes were proptosed, and both vitreous chambers were full of hemorrhage. Multiple fragments of bullet were revealed in the ethmoid bone by the roentgen rays. On recent examination the vitreous hemorrhages had cleared, leaving the right eye blind and with massive retinitis proliferans. The left fundus showed gross retinitis proliferans, but a little vision had been recovered in that eye. A callus from the injured right zygoma involved the right temporal muscle, limiting the opening of the mouth.

DISCUSSION

MR. CHARLES GOULDEN: I think the prognosis as regards sight is hopeless. I saw a number of somewhat similar cases in the World War, and the outlook was grave.

OPHTHALMOPLAGIC MIGRAINE. MR. S. R. GERSTMAN.

A woman aged 48 has been having recurring attacks of migraine ever since her fourteenth year. At the time of the first attack the right eye diverged, and it has not since returned to the normal position. In 1935 she had a severe attack and was hospitalized; she was found to have complete palsy of the third nerve on the right. Recent examination showed some weakness of muscles served by the third nerve. The ptosis had completely disappeared. With correction, vision was 6/12 in the right eye and 6/6 in the left.

NEOPLASTIC TYPE OF DISCIFORM DEGENERATION OF THE MACULA.
MR. ARNOLD SORSBY.

A woman aged 74 in 1931 had in the right eye with a plus 1 sphere vision of 6/6 and in the left eye vision of 1/60. The left central

area showed disciform degeneration of the "neoplastic" type, with hemorrhages. In October 1933 the left eye showed choroidal degeneration. In July 1935 vision of the right eye was less than 6/60. There were a hemorrhage into the vitreous and, in the left central area, an atrophic scar. In September 1936 the hemorrhages had disappeared from the right eye, and the swelling was more marked.

MACULAR DEGENERATION IN A PATIENT AGED TWENTY-FOUR. MR. FREDERICK T. RIDLEY.

A case of pure macular degeneration, beginning in the early twenties, was presented. Vision was not yet involved, although definite ophthalmic changes were noted. The mother, aged 52, with vision of 6/18, showed a similar condition. The condition was, therefore, hereditary, and was probably a dominant lesion.

Jan. 8, 1937

MR. W. H. McMULLEN, O.B.E., F.R.C.S., *President*

MELANOMA OF THE IRIS. MR. FRANK A. JULER.

A man aged 70 came for glasses. He had not noticed the discoloration of his iris. Examination revealed a tumor affecting two thirds of the width of the stroma of the iris and protruding forward into the anterior chamber, especially below the pupillary margin. There was a slight pulling of the pupil downward with slight ectropion uveae at the pupillary margin.

DISCUSSION

MR. CHARLES GOULDEN: A point of importance in diagnosing malignant disease is the deformity of the iris. The shape of the iris is not deformed by a nevus, but it is deformed by a malignant growth. In this case of Mr. Juler's, as in the last case of this condition that I observed, the root of the iris is free from growth. I successfully removed the tumor in my case by iridectomy, and there had been no recurrence several years after the removal.

MR. LEIGHTON DAVIES, Cardiff: I have observed two or three cases somewhat similar to this, but the patients were persons with dark irides. I have watched them for several years, and in each case there was an elevation of the tumor above the surface of the iris, without change in appearance for three or four years. My view about the condition in the patient now shown is that the growth is not malignant. If it is malignant, it is probably a melanotic sarcoma. If this is a case of melanotic sarcoma, I do not think that one can hope to save the patient's life by removing a portion of the iris.

MR. A. F. MACCALLAN: In the last few years I have shown, at meetings of the section, two patients with a condition similar to that of Mr. Juler's patient, and in them the condition has remained the same for many years. In my opinion, the growth in Mr. Juler's patient is a mole, not a malignant tumor.

MR. W. H. McMULLEN: The fact that the patient's wife had not noticed the defect of the iris suggests that it is not new. I think that the arrangement of the uveal pigment in this case is against a malignant growth. My advice would be to wait and from time to time measure the size of the pigmented mass.

HEMISPHERICAL FORMATIONS IN BOWMAN'S MEMBRANE. MR. EUGENE WOLFF and MR. T. KEITH LYLE.

A case of idiopathic epilepsy is presented in a stevedore aged 32. When 17 years old he began to suffer from dizzy spells, and at 21 years he had attacks of petit mal, followed at a later date by attacks of grand mal. Sometimes he has as many as three or four attacks a day. Examination showed that vision in the right eye was 6/6 and the fundus was normal. When 15 years of age he sustained an injury to his left eye with a tennis ball, and since then vision in the left eye has been blurred. At 23 years of age the sight of the left eye became much worse, a black film appearing to spread over that eye from the inner margin, and within three years he had become blind in that eye. The tension of the left eye was plus 2. The cornea was edematous, and the lens was slightly opaque. The optic disk was cupped; the media were hazy, and there was an extensive detachment of the retina. Treatment, which was carried out last year, was excision of the left eye, with the insertion into Tenon's capsule of a fat graft taken from the anterior part of the abdominal wall.

In regard to the pathologic aspect, abnormal, hemispherical and homogeneous structures were found in Bowman's membrane, occupying that portion which is next to the epithelium. These structures were actually in, not on, Bowman's membrane, and that membrane was not pushed backward, as was evidenced by its straight posterior border. The structures varied in size up to about 10 micromillimeters at their bases. Some sixty could be seen in each section, and there were more toward the central than toward the peripheral parts of the cornea. The hemispherical formations stained red with hematoxylin and eosin and orange-red or red with the Van Gieson stain. That the structures did not consist of amyloid was shown by the fact that they did not give the characteristic stain with methyl violet or congo red. It is probable they were hyaline, because they stained deep blue with Kühne's stain. Probably they were produced by degenerative products of the corneal epithelial cells or by degeneration of the perforating nerves.

EXPERIMENTAL STAINING OF THE RETINA DURING LIFE. MR. ARNOLD SORSBY, DR. A. ELKELES, DR. G. W. GOODHART and DR. I. B. MORRIS.

Our problem in this investigation was to determine whether we could make the translucent normal retina visible ophthalmoscopically by vital staining during life. Vital staining has been applied to other tissues, both for histologic purposes and as a therapeutic measure. An example of the latter is the use of various vital dyes in leprosy. The difficulties in vital staining of the retina are bound up with those which

have been encountered in staining the nervous system as a whole. There appears to be a barrier between blood and nerve tissue, a holding back of many substances circulating in the blood, preventing them from reaching the cerebrospinal fluid and the brain. The mechanism of this barrier has been the subject of much dispute. The blood-brain barrier prevents entry into the brain not only of various colloids which are found in the blood stream but also of toxins and such administered medicaments as arsenic. In this way the blood-brain barrier acts as a mechanism for the protection of the brain. This mechanism, however, is a detriment in some conditions, such as cerebrospinal syphilis, as it effectively prevents the therapeutic agent from coming into contact with the brain.

The existence of this blood-brain barrier has been studied through observing the action of various dyes which had been injected intravenously. Since the classic experiments carried out by Goldmann in 1913, it has been the prevailing view that the choroid plexus plays a dominant part in this mechanism, because trypan blue, the dye used in these experiments, was found mobilized in the plexus, while none of it was found in the brain tissue itself. The existence of the blood-brain barrier has been regarded as an insurmountable difficulty in vital staining of the brain. The same mechanism which prevents vital staining of the brain was presumably effective in preventing staining of the retina. Such work as has been done on vital staining of the eye has dealt largely with demonstrating the mechanism of the formation of the aqueous by the ciliary body. Recent work has shown that the blood-brain barrier is effective for acid dyes only, and basic dyes have been proved to penetrate the barrier and to stain the brain. Experiments carried out with acid dyes failed to produce any staining of the retina. The injection of basic dyes gave results which were difficult to interpret, since all the basic dyes are highly toxic and any staining that was obtained might well have been a supravital phenomenon, and held out little promise for clinical application.

Further work consisted of a search for a nontoxic dye which would pass the blood-brain barrier and produce the desired staining of the retina. Light green S. F. was found to answer the purpose, as it was nontoxic to the rabbit and produced transient staining of the fundus when injected intravenously. Staining of any duration clearly affecting the retina could not be obtained in the healthy rabbit. Degeneration of the rabbit's retina was then experimentally produced by the intravenous injection of septoniod, which is a solution containing sodium iodide, sodium iodate and iodine; when such rabbits were subjected to the injection of light green, the same transient staining as that noted in normal rabbits was obtained at the time of the injection. Half an hour later the fundus of these rabbits showed an intensive green coloration, which was of irregular distribution. This could not be obtained in the normal rabbit, and this failure was, presumably, because the normal rabbit's retina converts the dye into a leukobase by oxidation, the damaged retina doing so to a much more limited extent. Further investigations have shown that the same results can be obtained by intramuscular injection of the dye. The histologic investigations showed that the dye was situated in the retina.

DISCUSSION

MR. EUGENE WOLFF: Some time ago there appeared in the ARCHIVES an article reporting a series of cases of some cutaneous disease in which the fundus was stained yellow, and in those cases, I think, it was decided that the dye was in the vitreous. The impression given in those cases was that the observer was seeing the retina through the dye in the vitreous. It is difficult to exclude that possibility, and I ask whether any of these animals referred to by Mr. Sorsby. were looked at with the eye opened and the vitreous removed.

MR. ARNOLD SORSBY: Mr. Wolff is referring to a case in particular which was published in the September 1936 issue of the ARCHIVES (Gerber, A., and Lambert, R. K.: Blue Appearance of the Fundus Caused by Prolonged Ingestion of Methylthionine Chloride, ARCH. OPHTH. 16: 443 [Sept.] 1936), accompanied by a colored plate showing a blue fundus. Four patients with urogenital tuberculosis were given methylthionine chloride (methylene blue) for four years; the fundus assumed a blue color. That the coloration was due to staining of the vitreous was a conjecture and not a fact. My collaborators and I have opened a number of eyes; we found that the vitreous was always unstained, the lens was always unstained and the aqueous was sometimes stained and sometimes not stained. The problem is to find a group of dyes which get past the choroid plexus and reach the brain, i. e., its nervous tissue, which dyes shall be nontoxic and shall not cause local damage.

NEW YORK ACADEMY OF MEDICINE,
SECTION OF OPHTHALMOLOGY

Dec. 21, 1936

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

A CASE OF OXYCEPHALY. DR. GIROLAMO BONACCOLTO.

A boy aged 13, with a negative family history, born at the end of a full term pregnancy, presented an abnormal skull which was classified as oxycephalous. The following signs were found: a characteristic roentgen appearance of convolution depressions on the walls of the skull and shortening of the anteroposterior diameter of the orbit; exophthalmos of 27 mm.; divergent strabismus; primary atrophy of the optic nerve with loss of vision in both eyes and a corresponding contraction in the visual fields. Accompanying the report was a review of the literature.

MALIGNANT MELANOTIC TUMOR OF THE LIMBUS. DR. ROWLAND H. MERRILL.

A case was demonstrated of malignant pigmented tumor of the limbus occurring in a white woman of 30. A large pigmented non-

movable nodule was present at the temporal limbus of the right eye. A highly vascular, succulent, infiltrative mass extended from the nodule around the limbus for half its circumference and invaded the superficial layers of the cornea in a band about 3 mm. wide. A small chain of metastatic nodules occurred in the conjunctiva 5 mm. below the cornea. Vision in each eye was 20/70, and was corrected to 20/30 +. The tension was normal. Both lenses showed coronary cataracts. The fundi were normal.

DISCUSSION

DR. ALGERNON B. REESE: I doubt whether enucleation will prove sufficient in this case, because it seems to me there is extensive involvement of the conjunctiva. If all the involved conjunctiva were removed at the time of the enucleation, I do not believe there would be a sufficient amount left to cover the socket. In the cases I have observed, as a rule, there is rather extensive involvement of the basal layer of the conjunctiva, even farther away than the obviously involved areas. Therefore I believe that exenteration is the procedure indicated.

KERATOPLASTY AND IMPROVED INSTRUMENTS. DR. R. TOWNLEY PATON.

Rabbits' eyes afford the most economical and best material for experimentation. Metal stencils or a double-bladed knife with angulated blades may be used to outline grafts in certain cases. For eyes with a shallow anterior chamber and iris bombé, a broad double-edged knife, concave on one surface, may be found advantageous.

DISCUSSION

DR. RAMON CASTROVIEJO: Most of the ophthalmologists who have had any experience in making corneal transplants have been more interested in reporting success than in finding out causes for failure and ways to prevent this failure. It is difficult to state at the present time which of the methods of corneal transplantation now used is the best. It may happen, as in any other field of surgery, that the operator accustomed to use a particular method will obtain with this method better results than if he were to use other methods with which he has had less experience. Therefore, I am sure that Elschmig, using the old von Hippel technic of corneal transplantation, will have better results than if he were to use Thomas', Filatov's or my method, and vice versa.

At the Institute of Ophthalmology of the Columbia-Presbyterian Medical Center seventy corneal transplantations have been performed. After this series of operations it was possible to tell beforehand those cases in which the condition was favorable for corneal transplantation and those in which it was not. The results obtained in the last five months have corresponded with the preoperative diagnosis. As a rule, in the cases in which the condition was diagnosed as unfavorable the transplant became either nebulous or opaque. Of the cases in which the condition was diagnosed as favorable, in more than 90 per cent the transplant remained permanently transparent.

Dr. Paton has mentioned the difficulty in diagnosing anterior and posterior synechiae when the cornea is densely opacified. This problem has been taken up by some authors. Thomas and I have described

methods of transillumination which render easy the diagnosis of anterior and posterior synechiae in cases of very dense leukoma.

Dr. Paton has also said that in some instances, especially when the knife is dull, he uses the device that he has shown tonight. This device when applied to the cornea makes it easy to outline in this structure either a square or a triangle. It should be remembered that before operation for corneal transplantation the knife should be tested to make sure that it is in good working condition. No one would think of starting an operation for cataract before making sure that the knife used to make the incision was sharp. The incisions made to outline the flap to be transplanted should be clean. Therefore the knife must be sharp; otherwise, irregular edges of the transplant and cornea of the host will be obtained which may defeat the success of the operation.

To outline a flap my associates and I use a knife with blades made from razor blade material. It is very sharp and is generally used in no more than two or three operations. The knife is tested before operation in the same manner as the knife used to make incisions for operations for cataract.

Dr. Paton has shown some diagrams illustrating a knife about 4 mm. in width, which goes through the anterior chamber. The puncture seems to be located in the cornea about 2 mm. from the limbus, and the counter-puncture is at the same distance from the limbus on the opposite side. With this knife, Dr. Paton stated, injury of the iris and lens may be prevented during dissection of the corneal leukoma. The idea expressed by Dr. Paton has been worked out practically by Dr. Filatov, who, after making the puncture and counter-puncture as described by Dr. Paton, introduces into the anterior chamber an ivory plate that he calls a prophylactic spatula. Dr. Filatov has been using this prophylactic spatula for some years and reported a number of successful results with it, but recently I have been told that he is no longer using the prophylactic spatula because the combination of knife and spatula caused, in some instances, injury of the iris and lens, which is the very thing that he was trying to prevent.

Dr. Paton has presented a double-bladed knife with the blades placed at a certain angle. He claims that shelving may be obtained with this while one is outlining the corneal flaps. It is difficult to understand how with a double-bladed knife in which the distance between the blades does not vary while the incision is made it is possible to obtain a shelving. The shelving claimed to be obtained by Dr. Paton is a mechanical impossibility.

For the past two years my associates and I have been experimenting with a new type of trephine made of a suction spoon which fastens to the cornea and a rotating blade placed at an angle of from 5 to 45 degrees which, at the same time that it rotates around the suction spoon, penetrates deeper and deeper. So far two such trephines have been completed. The last one worked satisfactorily in normal corneas but did not perform well when the resistance of the cornea had been increased by proliferation of connective tissue. More work is being done to overcome the difficulties encountered, and I am presenting the idea in the hope that some of the members might aid in finding the solution. It is gratifying to know that more persons every day are interested in keratoplasty, a problem which, when solved, will benefit so many unfortunate ones.

DR. R. TOWNLEY PATON: Dr. Castroviejo has given this subject a great deal of thought, but I believe that he forgets the important fact that there is considerable upward pressure of the cornea against the twin-bladed knife, and a slight bulge is produced, part of which is compensated for by the angulation of the blades. The operator should on no account attempt to penetrate through the entire thickness of the cornea with this knife. Another advantage of the double-bladed knife which I forgot to mention is that a more clearly outlined window may be obtained with this instrument, owing to the slight tearing action of the angulated blades as they are passed over the epithelial surface of the cornea.

THE DIAGNOSTIC SIGNIFICANCE OF EPINEPHRINE WHEN APPLIED BY
INSTILLATION INTO THE CONJUNCTIVAL SACS IN THE STRENGTH
OF 1:1,000. DR. LOUIS HUBERT.

This article will be published in full in a later issue of the ARCHIVES.

CORRECTION OF COMBINED VERTICAL AND LATERAL STRABISMUS. DR.
CHARLES E. DAVIES.

A case was presented in which there was complete correction of esotropia of 75 centradis associated with hyperphoria of 3 centradis in the primary position and hypertropia of 26 centradis on looking up and to the left by a single recession of the right internal rectus muscle and resection tenotomy of the right inferior oblique muscle.

DISCUSSION

DR. JAMES WATSON WHITE: Dr. Davies has shown an excellent result of an operation for combined vertical and lateral strabismus.

There is one phase of this question which Dr. Davies had no cause to mention but which I have failed to emphasize enough. When the eye that has the paretic superior rectus muscle is used to fixate, the inferior oblique muscle has a secondary deviation which usually results in hypertrophy. In this case tenotomy of the inferior oblique muscle was indicated. However, when the nonparetic eye is used to fixate, no hypertrophy develops, as constant overstimulation is not present. When this condition is present, resection of the paretic superior rectus muscle is indicated.

OPERATION FOR RETINAL DETACHMENT. DR. JOSEPH LEVINE.

It is suggested that in localizing the retinal tear the ora serrata be used as the fixed point and not the optic disk, because the distance from the limbus to the optic disk depends on whether the temporal or the nasal side of the globe is measured. A curved millimeter rule is recommended, as advocated by Vogt, in measuring distances on the globe. To set up adhesive choroiditis an electrical current (a combination of cutting current and coagulation current) of a wavelength of 300 meters is used. Between 30 and 40 milliamperes are applied through individual steel pins which are inserted into the globe about 4 mm. apart. A barrage is made around the tear and also over the region of the detachment. A small opening is made with the trephine over the most

pendulous portion of the detachment, and the choroid is punctured to allow drainage of the subretinal fluid. The individual pins are then removed.

SUMMARY OF ORTHOPTIC WORK AT THE MANHATTAN EYE, EAR AND THROAT HOSPITAL. DR. FRANK CONRAD KEIL.

Approximately 500 patients with strabismus were examined in the orthoptic clinic of the Manhattan Eye, Ear and Throat Hospital during the past two years. This summary is in no respect a record of research work. Each case was assigned to one of the following groups:

Group	Type of Strabismus	Number of Cases
A	Monolateral convergent concomitant.....	377
B	Alternating convergent concomitant.....	70
C	Divergent concomitant	85

GROUP A.—After wearing glasses from two to three weeks, 99 patients had fusion at orthophoria, and 17 had fusion and normal adduction but limited abduction. Fourteen with gradual reduction showed second degree fusion after from three to four months. With glasses, all had at least second degree fusion with occasional stereopsis, normal duction and no suppression, amblyopia or abnormal retinal correspondence. Without glasses, esophoria of from 10 to 40 degrees was present. Of 51 patients who had orthoptic training (given for one-half hour periods twice a week over a period of from six to twelve months), 11 could fuse at orthophoria; 4 fused with prisms base out of 10 degrees, and 1 had unstable fusion. Three had improved fusion and amplitude, but high esophoria remained with glasses. There were 14 who had doubtful fusion and required prisms base out for binocular vision. Thirty-three made too few visits or were uncooperative.

GROUP B.—Ten patients with deviation of less than 10 degrees had second degree fusion with glasses. Eleven with deviation of 40 degrees or more received preoperative training. Of the 20 who had postoperative orthoptic training, 9 had unstable second degree fusion, although 7 of these had preoperative training. Five whose vision was slightly overcorrected fused after training, and the remainder, some of whom had overcorrected vision, and others, undercorrected vision, had no binocular vision. Twenty-four failed to cooperate, and 2 had alternation of fixation.

GROUP C.—Seventy of the 85 patients were hyperopic. Only those with marked exotropia applied for treatment. The slight exophoria characteristic of myopia was probably improved with glasses. Seventeen persons with divergence excess of from 6 to 30 degrees had improved tolerance for fusion after orthoptic training. Eighteen who had vision characterized by high deviation, associated hyperphoria, little or no binocular perception, remote near point of convergence, which were frequently periodic, and cosmetic defects, were not aided by orthoptics. Five who had postoperative training had good fusion. The remaining patients who had postoperative training and who had under correction or overcorrection showed no improvement.

An orthoptic department may be considered as a "clearing house" for cases of strabismus. It has a major place in the education of parents in respect to the treatment of the "cross-eyed child." Its follow-up has been valuable in maintaining the treatment of amblyopia. The results of preoperative and postoperative orthoptic treatment have been encouraging, and the painstaking procedure used in examination has been helpful in guiding the surgeon to a more perfect solution of a difficult problem.

DISCUSSION

DR. LEWIS WEBB CRIGLER: The essentials of the treatment of strabismus are as follows:

1. A child with strabismus must be brought to the ophthalmologist as soon as the squint is detected.
2. The refractive error must be carefully and fully corrected.
3. Treatment must be begun at once by excluding vision from the fixing eye.

Nothing will be accomplished by muscular exercises or fusion training if vision in the squinting eye is not materially improved.

It is a waste of time to attempt to correct squint by orthoptic methods in cases in which there is a complete absence of the fusion sense, and in the vast majority of cases squint in time will be relieved merely by the use of proper glasses, provided the fusion sense is present. The sphere of usefulness of the synoptophore and similar instruments is in those cases in which the fusion sense has been suppressed but is not entirely lacking. I question the efficacy of such treatment unless it is carried on more intensively than it is now practiced, but the stumbling block immediately arises in the failure to obtain proper cooperation.

The whole subject is a perplexing one the answer to which has not yet been completely solved. Good has been accomplished in that ophthalmologists have been aroused to a greater sense of obligation to the squinting child. The chief advance that has been made is that one is better able to separate the surgical from the nonsurgical cases.

DR. JOSEPH I. PASCAL: I think it is good practice to start fusion training at the earliest possible time, even when vision in the squinting eye is only 20/200. Vision of 20/200 means the ability to recognize an object which subtends an angle of about 50 minutes. Most objects in the child's environment—his playthings and eating utensils, for example—and, for that matter, things in the adult's surroundings, subtend an angle of 50 minutes or more. These objects are recognizable by the amblyopic eye, and there is no reason why the eyes should not be trained to fuse the recognizable images, if there is any fusion faculty capable of being developed. As a matter of fact, trying to develop fusion from the beginning, say, as soon as vision is 20/200 in the squinting eye, helps to develop the vision of that eye. The very procedure used in trying to build up the working together of both eyes has a beneficial effect on the amblyopic eye.

There is one other point I wish to touch on. Dr. Keil in his report mentioned few cases of so-called false projection and abnormal retinal correspondence. These are phases of orthoptic training that should be thoroughly investigated. Abnormal retinal correspondence, as the term is now generally used, means primarily the habit of fusing the macular

image of the fixing eye with the nonmacular image of the deviating eye. Dr. Pugh in her recent book "Squint Training" stated that about 50 per cent of squinting patients have abnormal retinal correspondence. Worth, after a study of 2,000 cases, said that such a condition is rare. He called the nonmacular portion of the retina, the image of which is projected so that it would be fused with the macular image of the fixing eye, a "false macula." This term is now used differently. But the important fact is that he found this condition to be rare, though he found what he termed false projection to be common. Furthermore, this condition, which is really abnormal binocular vision (the fusion of a macular image with a nonmacular image) is scarcely mentioned by the French writers, notably by Drs. Cantonnet and Filliozat in their latest book "Strabismus," and they also have studied an enormous number of cases. The presence of such an extraordinary variance among observers suggests the need for a careful checking up on the condition.

MISS DOROTHY SHADD: As to requiring visual acuity of 20/70 for the beginning of training, I agree that it is possible with large objects to get fusion with much lower visual acuity than that with which one can get fusion when small objects are used; one can get fair fusion with vision of 20/20 in one eye and of 20/200 in the other, but there is apt to be frequent suppression of vision in the amblyopic eye. That is the reason I have taken visual acuity of 20/70 as the requirement for duction training.

My associates and I observe a large number of persons with abnormal retinal correspondence among those seen in the orthoptic clinic, but their records do not appear in our slides because these patients are seldom in the groups who show normal fusion or duction. They do not easily acquire normal fusion or duction. In general, I find that abnormal retinal correspondence occurs in the group with monolateral convergent strabismus in conjunction with definite amblyopia.

DR. JOHN M. WHEELER: May I ask what is meant by duction?

DR. FRANK CONRAD KEIL: Duction is a term used in orthoptic training to indicate the action of fusion. Its extent is measured by the amplitude of fusion with prisms base in or base out.

LESIONS OF THE FUNDUS IN POLYCYTHEMIA. DR. MARTIN COHEN.

This article will be published in full in a later issue of the ARCHIVES.

Book Reviews

Wills Hospital Eye Manual for Nurses. By Gladys E. Cole, R. N., Chief Nurse, Wills Eye Hospital. Price, \$1.75. Pp. 202, with 97 illustrations. Philadelphia, W. B. Saunders Company, 1936.

Miss Cole aims in this small manual to acquaint nurses with what they should know about the eye in practicing ophthalmic nursing. The book contains chapters on the anatomy of the eye and a short description of the diseases of the eye which have been observed by ophthalmologists of the Wills Hospital.

The important parts which deal directly with the nurses' activities are grouped under the following headings: irrigation of the eyes and the instillation of drops; examination and treatment of patients with ocular disorders; dressings, bandages and shields for patients with ocular disorders; preparation for operation; instruments for operation on the eye, and the procedure of operation. The nursing care in cases of cataract is found in a chapter giving the history of the operation for cataract.

The directions for treatment are clearly outlined and will be useful to the ophthalmic nurse, particularly as the procedures enumerated are those that have stood the test of time in that excellent institution the Wills Hospital of Philadelphia.

ARNOLD KNAPP.

Medical Classics. Edited by E. C. Kelly, M.D., Albany Medical College, Albany, N. Y. In volumes of 10 numbers. Price, \$10 per volume. Pp. about 1,000. Baltimore: Williams & Wilkins Company, 1936.

This publication aims to awaken the interest of all medical workers in the historical side of their profession. The important papers of previous great masters of medicine are printed in full, and, in addition, a brief life of the author, a bibliography, a brief sketch of the subject treated and the best available portrait of the author are given.

The subject chosen for the first number is Sir James Paget and his papers on osteitis deformans, Paget's disease of the bones, and his paper on Paget's disease of the nipple.

The subjects for the succeeding numbers are excellently chosen, and the publication deserves an enthusiastic reception. The importance of reading the original papers of the great masters cannot be overemphasized.

"Medical Classics" is dedicated to Dr. Joseph Lewi Donhauser. The editor welcomes suggestions concerning future subjects.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.

President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6^e).

Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ost-flandern, Belgium.

All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.

Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.

Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.

Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.

Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.

Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.

Secretary: T. Harrison Butler, 81, Edmund St., Birmingham, England.

Place: Birmingham and Midland Eye Hospital.

Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.

Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.

Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.

Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Time: June 11, 1937.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON
OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.

Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.

Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

Place: Hot Springs, Va. Time: June 3-5, 1937.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.

Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.

Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
 Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
 Place: Salt Lake City, Utah. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
 Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
 Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
 Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
 Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
 Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
 Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
 Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
 Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
 Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
 Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
 Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
 Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
 Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
 Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.

Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.

Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.

Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.

Place: Des Moines.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.

Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.

Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.

Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.

Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.

Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.

Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.

Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.

Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.

Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.

Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
EYE, EAR, NOSE AND THROAT

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.

Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.

Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.

Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.

Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.

Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.

Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.

Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.

Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.

Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.

Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
 Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
 Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
 Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
 Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
 Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
 Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
 Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
 Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
 Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
 Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
 Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
 Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
 Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
 Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
 Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
 Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
 Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
 Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.

Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.

Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.

Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.

Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.

Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.

Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in alphabetical order.

Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.

Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.

Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.

Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.

Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.

Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.

Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
 Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
 Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
 Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St. New York.
 Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
 OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
 Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
 Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
 Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
 Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
 Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
 Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
 Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
 Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
 Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
 Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.

Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.

Place: Oscar Johnson Institute Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.

Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.

Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.

Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.

Place: Society's Building, 2180 Washington St., San Francisco.

Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.

Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.

Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.

Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.

Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.

Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W. Toronto.

Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.

Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.

Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

DEFECTS IN VISUAL FIELD OF ONE EYE ONLY IN PATIENTS WITH A LESION OF ONE OPTIC RADIATION

MORRIS B. BENDER, M.D.

AND

ISRAEL STRAUSS, M.D.

NEW YORK

Lesions of the optic radiation cause contralateral homonymous defects in the fields of vision. The defect may be quadrantic or hemianopic, with or without sparing of central vision. Sometimes, however, careful perimetric determinations reveal partial and unpaired blind areas, i. e., a defect in one section of the field of vision of one eye but not in the corresponding field of the other eye. The commonest type of unpaired defect in cases of lesions of the optic radiation is known as the temporal crescent or half moon defect. The terms temporal crescent and half moon have reference to either one of the two areas which are temporal and peripheral to the binocular field of vision (fig. 1). Each of these areas is projected on the retina of one eye only (on the nasal retina) and therefore represents the uniocular field of vision. It corresponds to the phylogenetically oldest monocular vision in primates.

ANATOMY OF THE FIBERS OF THE TEMPORAL CRESCENT IN THE VISUAL PATHWAYS

Brouwer and Zeeman¹ demonstrated that after destruction of the peripheral portion of the nasal retina there was degeneration of a bundle of fibers located in the median portion of the optic nerve that crossed in the optic chiasm to the median part of the optic tract and spread downward ventrally into the lateral geniculate body (fig. 1). Wilbrand² showed that this small bundle was located in the ventrolateral portion of the optic chiasm and seemed to contain only crossed fibers.

From the Neurologic Service of Dr. Israel Strauss, the Mount Sinai Hospital.

1. Brouwer, B., and Zeeman, W. P. C.: The Projection of the Retina in the Primary Optic Neuron in Monkeys, *Brain* **49**:1, 1926.

2. Wilbrand, H.: Schema des Verlaufs der Sehnervenfasern durch das Chiasma, *Ztschr. f. Augenh.* **59**:135, 1926.

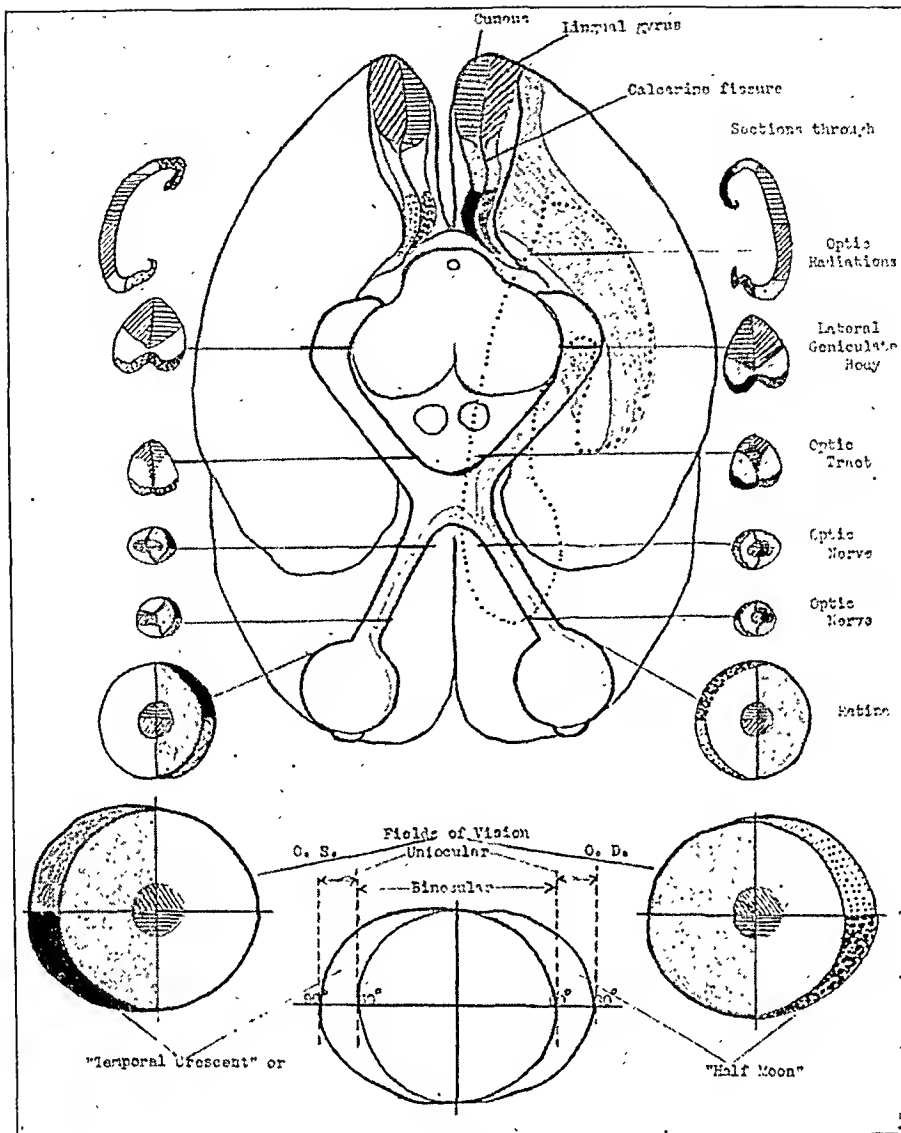


Fig. 1.—Schema illustrating the probable course of the afferent visual pathways at different levels from the retina to the occipital cortex and their projected visual fields. Solid black represents the inferior, and the lighter shade the superior, temporal crescent of the left field of vision; the heavy dots represent the inferior, and the lighter dots the superior, temporal crescent of the right field of vision. These are projected from the corresponding points of the nasal retina of each eye. From the nasal retina the fibers may be traced through the mesial part to the optic nerve, crossing completely in the chiasm to the ventral portion of the opposite optic tract and lateral geniculate body, the mesial portion conducting the fibers for the superior, and the lateral portion the fibers for the inferior, part of the crescent. In the optic radiation the fibers for the superior, and those for the inferior, portion of the crescent separate and course along their respective quadrants at opposite edges of the radiation. The fibers come together again in the most anterior part of the area striata, the fibers for the superior part of the crescent terminating in the cortex above, and those for the inferior part of the crescent in the cortex below, the calcarine fissure. The solid shaded areas represent the binocular left homonymous field of vision. The horizontal lines represent the lower macular, and the diagonal lines the upper macular, field of vision.

The course of this bundle of fibers beyond the lateral geniculate body was ascertained by Poliak³ in 1932. Pfeiffer⁴ in 1925 suggested that these fibers were located in the ventral rim of the optic radiation. Traquair⁵ and Wilbrand² opined that the fibers for the crescent occupied the most mesial portions of the radiations. While Pfeiffer believed that all the fibers reached the occipital cortex by passing the posterior horn from below, Poliak³ found that they reached the cortex from above and below (fig. 1). In the cortex of the occipital lobe, the most anterior portion of the area striata harbors the projected crescent of the "cortical retina," the upper half of the crescent being located in the upper lip, and the lower half of the crescent being located in the lower lip, of the most anterior portion of the calcarine cortex. Working with a chimpanzee, Spence and Fulton⁶ extirpated the entire left occipital lobe and at a later date resected part of the right occipital lobe. Subsequently it was observed that the chimpanzee had vision in only the periphery of the left homonymous field. When the brain was finally examined it was seen that only the anterior portion of the right area striata was intact. These investigators concluded that their experiments supported the view that the anterior portion of the area striata around the calcarine fissure is the cortical terminus for peripheral vision.

Kronfeld⁷ and Soriano⁸ reviewed the cases reported in the literature. The etiology was varied. The best anatomic contributions were derived from the subjects with cerebral injury. In dealing with cases of tumor of the brain one cannot disregard the possibility of edema and other distant reactive phenomena as causal factors in disturbance in the field of vision, so that no exact anatomic information may be available. On the other hand, a good deal of pathologic physiology may be learned from tumor material. For example, when a tumor grows in or near the area occupied by the optic radiation, there results usually progressive alteration of perception in the contralateral field of vision; the change in the function of vision is gradual, although usually fluctuating. A study of this gradual change may reveal knowledge which could not be ascertained by other methods.

3. Poliak, S.: *The Main Afferent Fibre Systems of the Cerebral Cortex in Primates*, Berkeley, Calif., University of California Press, 1932, vol. 2, p. 370.

4. Pfeiffer, R. A.: *Myelogenetische Untersuchungen ueber den zentralen Abschnitt der Sehleitung*, in Foerster, O., and Wilmanns, K.: *Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie*, Berlin, Julius Springer, 1925.

5. Traquair, H. M.: *An Introduction to Clinical Perimetry*, St. Louis, C. V. Mosby Company, 1927, p. 211.

6. Spence, K. W., and Fulton, J. F.: *The Effects of Occipital Lobectomy on Vision in the Chimpanzee*, *Brain* **59**:35, 1936.

7. Kronfeld, T.: *The Temporal Half Moon*, *Tr. Am. Ophth. Soc.* **30**:431, 1932.

8. Soriano, F. J.: *Semiología de las hemianopsias homónimas*, *Semana méd.* **1**:1005, 1931.

The exact incidence of occurrence of peripheral temporal crescentic defect is difficult to determine, because the visual fields could not always be determined perimetrically. Of one hundred patients with verified tumor implicating the optic radiations for whom the fields of vision were charted, ten showed defects of the temporal crescentic variety. The types of unpaired defects found in the peripheral temporal field of vision are illustrated and discussed in the following case reports.

REPORT OF CASES

HEMICRESCENTIC DEFECTS

CASE 1.—S. F., a boy aged 12, complained of occasional attacks of headache and nausea for six weeks prior to admission to the hospital; for twelve days he experienced twitchings of the right side of the body.

Examination revealed faciobrachial paresis on the right, with a concomitant sign of involvement of the pyramidal tract. The fundi were normal. Central visual acuity was 20/20 in each eye. Repeated studies showed a well defined hemi-

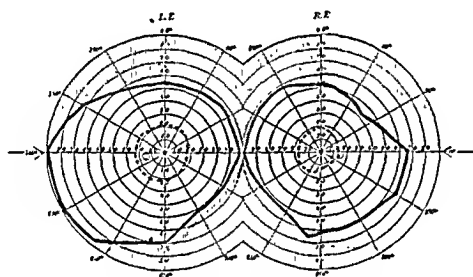


Fig. 2 (case 1).—Visual acuity was 20/20 in each eye. The fundi were normal. A crescentic defect in the upper and, to a lesser extent, in the lower right temporal field was revealed on examination with test objects 4 and 2 mm. in size.

All the observations of the visual fields in the studies reported were made with the Ferree-Rand perimeter. The test objects, the size of which is indicated in each case, were in motion during the examination. Daylight illumination was used. In the charts of the visual field the solid lines represent the border of the field for white objects, and the broken lines the border of the field for colored objects.

crescentic defect in the periphery of the upper and, to some extent, of the lower temporal quadrant of the right field of vision. The field for color was concentrically contracted in the right eye. The spinal fluid pressure was 120 mm. of water. Nine days after the patient's admission an encephalogram revealed normal ventricular and subarachnoid systems. Five days later a tumor (spongioblastoma multiforme) was removed at operation from the left paracentral lobe.

Comment.—In this case there was no evidence from the appearance of the fundus oculi, encephalogram and lumbar puncture to indicate the presence of distant pressure phenomena or distortion of cerebral or optic nerve structures to account for the hemicrescentic and the cres-

centric defect in the peripheral temporal field of vision. Since the pathologic signs were those of a malignant infiltrating tumor, it is possible that the defect of the field was the first manifestation that a part of the optic radiation conducting the fibers for the crescent was implicated by tumor tissue or by reaction around the neoplasm.

CASE 2.—A. F., a school teacher and housewife aged 48, complained that for four and one half years before admission to the hospital she experienced auditory hallucinations (heard her own voice but could not describe the words); for four years she had spells during which she imagined she saw objects; for two years she had disturbances of memory and attacks of petit mal.

Examination showed obesity, irregular pupils which reacted poorly to light and in accommodation, blurring of the margins of the optic disks and paresis of the supranuclear part of the facial nerve on the right. Though usually cheerful and somewhat facetious, she was irritable on occasions. She had slight difficulty in naming common objects.

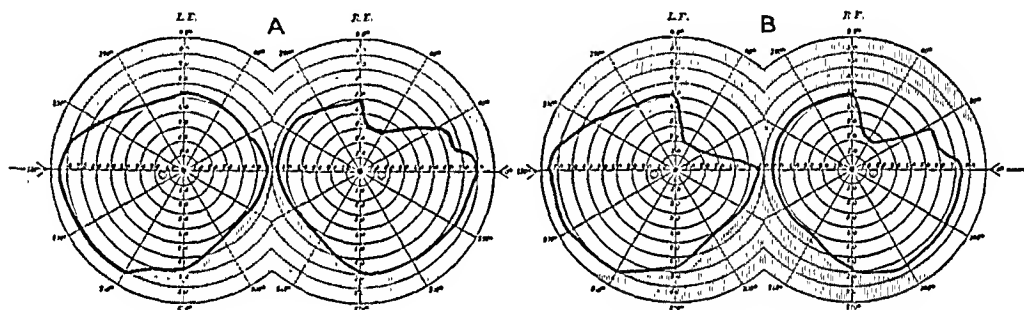


Fig. 3 (case 2).—There were complex hallucinations in the right field of vision. Central visual acuity was 20/20 in each eye. The fundus of each eye showed blurring of the edges of the disk. On examination with a test object 1 mm. in size a hemicrescentic defect was found in the right upper peripheral temporal field of vision (A), which when it became larger resulted in a homonymous field defect (B).

Four days after admission she became cooperative. A perimetric examination of the field, on July 6, 1935, consistently showed a hemicrescentic defect in the periphery of the right upper temporal field of vision (fig. 3 A). Central visual acuity was 20/20 in each eye. Two days later she complained that she saw figures which appeared to be close to the eye in the right field of vision. On the same afternoon repeated visual studies indicated homonymous superior quadrantanopia in the right field, which was incomplete and incongruent (fig. 3 B).

The patient refused operation. Three months later papilledema with hemorrhages developed bilaterally. At that time she was too uncooperative for studies of the visual fields to be made. Operation disclosed a huge meningioma located deep in the left sylvian fissure.

Comment.—In this case the unpaired hemicrescentic scotoma was a forerunner of incomplete homonymous superior quadrantanopia in the right field. It appears that as soon as the temporal defect in the right eye had reached a certain size the paired nasal quadrant in the other eye

became similarly affected. Interesting was the complex visual hallucination in the right field of vision which the patient experienced just before the perimetric examination.

CASE 3.^o—J. K., a man aged 44, entered the hospital with the chief complaint of "bandlike headache." Four years before admission he had an attack of unconsciousness. Four and a half months prior to admission he suffered from headache and tremor of both hands. Two weeks later he began to vomit. Three weeks before entering the hospital he became unsteady in his gait and had weakness in the right arm and face; his vision became blurred, and the headaches felt more severe—"like a band around the left temple." During the last two weeks he had diplopia, polydipsia and drowsiness.

Examination revealed hemiparesis and diminished sensibility on the left side of the body and signs of involvement of the pyramidal tract bilaterally. All the ocular movements, including those of convergence, were slightly impaired. Both external rectus muscles were paretic. The fundi revealed papilledema, hemorrhages and exudates. The patient was drowsy and somewhat euphoric. The spinal fluid was under an initial pressure of 340 mm. of water.

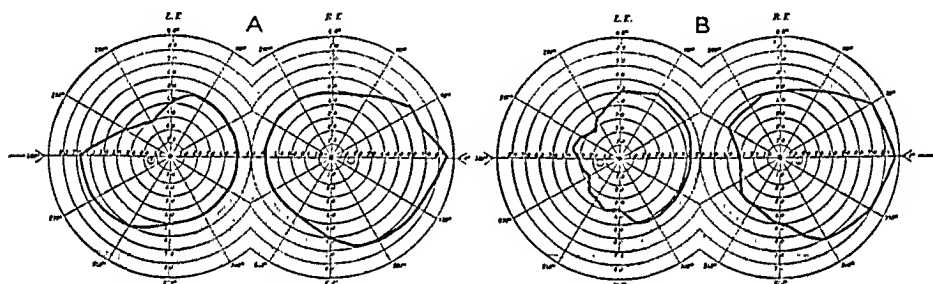


Fig. 4 (case 3).—There was blurring of vision. Central visual acuity was 20/50 in each eye. The fundus of each eye showed papilledema. There was a crescentic defect in the left peripheral temporal field of vision, which was most pronounced in the upper crescent (A). Ten days later, the field defect progressed to incomplete homonymous hemianopia, involving the binocular paracentral fields (B). Examination was made with a test object 4 mm. in size.

On March 18, 1932, the day following admission to the hospital, perimetric tests indicated a well defined hemispherical in the left upper, and a narrow hemispherical in the left lower, peripheral temporal field of vision (fig. 4 A). Central visual acuity in each eye was 20/50.

Three days later a ventriculogram taken after trephining in the left occipital region indicated what was interpreted to be a neoplasm compressing the right

9. The visual fields and observations at autopsy in this case were previously described by Globus and Silverstone (*Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances*, *Arch. Ophth.* **14**:325-386 [Sept.] 1935). In connection with this case they called attention to the fact that in homonymous lateral anopias "the defect on the side opposite the lesion was almost always greater than that on the same side. . . . An attempt to correlate these findings with the extent of the lesion found on postmortem examination did not yield reliable substantiating observations."

lateral ventricle from the midline and from below. Under observation partial ophthalmoplegia developed on the right (with ptosis of the upper lid, paresis of the superior, internal and external rectus muscles and dilation and fixation of the pupil). Craniotomy was postponed because of a preexisting infection of the scalp (a furuncle).

Perimetric examination, repeated ten days after the first test, on March 28, showed a total crescentic defect in the periphery of the left temporal field and a narrow defect in the periphery of the right nasal field of vision, so that the picture was one of incomplete asymmetrical homonymous hemianopia in the left field (fig. 4B). The infection of the scalp did not clear, and the patient died three weeks later from complicating erysipelas.

Autopsy disclosed a large cystic tumor (spongioblastoma multiforme), which seemed to have originated in the region of the right hippocampal gyrus. It infiltrated the inferior half of the right temporal lobe and the anterior portion of the occipital lobe. The corpus callosum and the ventricular system were displaced to the left.

Comment.—In this case the earliest defect in the field of vision was closely similar to the type found in the two previous cases, yet the

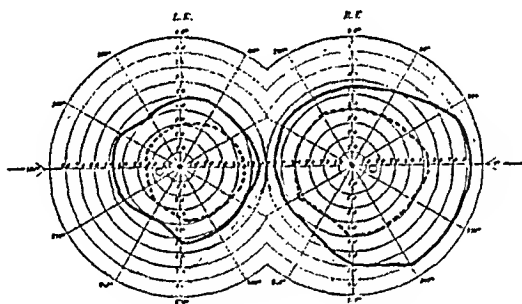


Fig. 5 (case 4).—There were crude visual hallucinations and blurred vision. Central visual acuity was 15/30 in each eye. The fundus of each eye showed papilledema. On examination with a test object 4 mm. in size a total crescentic defect for form and color was found in the left peripheral temporal field of vision.

location of the neoplasm was different in each case. Another feature was the incongruity of the defects in the fields of vision, the defect being more marked in the temporal than in the homonymous nasal field of vision.

PERIPHERAL TEMPORAL TOTAL CRESCENTIC SCOTOMA

CASE 4.—J. S., a man aged 43, was readmitted to the Mount Sinai Hospital because of headache. After he first entered the hospital, ten months previously, he had a partial pneumonectomy and partial excision of the wall of the chest and the diaphragm for carcinoma of the right lung with invasion of the wall of the chest and the diaphragm. Six weeks prior to the second admission he had severe headache in the occipital and the right frontal regions and blurring of vision. Occasionally he saw streaks of light with his eyes closed. Distinct changes in personality were noted by his relatives.

Examination revealed bilateral early papilledema and a crescentic defect for both form and color in the left peripheral temporal field of vision (fig. 5). Central visual acuity of each eye was 15/30. There was paresis of the left central portion of the face and abdominal reflexes were absent bilaterally. Under observation he became drowsy, and the pulse rate dropped to 52 beats per minute. Lumbar puncture showed a pressure of 180 mm. of water, a total protein content of 175 mg. per hundred cubic centimeters and 4 cells per cubic millimeter. Exploratory craniotomy on the right gave negative results. The patient fared poorly and died two days later. Autopsy revealed edema of the right hemisphere, with displacement of the ventricular system to the left. On the ventral and mesial surface of the anterior portion of the occipital lobe and the posterior portion of the temporal lobe was a tumor measuring 3 cm. in the lateral diameter. The surrounding area was gray and soft. The tumor nodule had metastasized from a carcinomatous focus in the bronchus. There were no other metastases to the brain.

Comment.—In this case the defect in the field of vision illustrates the typical total crescentic defect in the peripheral temporal field.

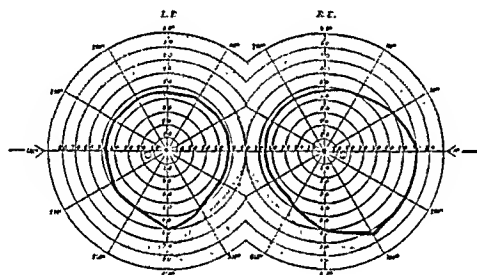


Fig. 6 (case 5).—Central visual acuity was 10/15 in the right eye and 10/30 in the left. The fundus of each eye showed blurring of the margins of the disk. On examination with test objects 4 and 2 mm. in size a total crescentic defect was found in the left peripheral temporal field of vision.

Although the neoplasm was well localized, there was too much surrounding edema to enable one to draw any definite conclusions as to the location of the fibers conducting vision from the periphery of the nasal retina through the optic radiation. The lesion implicated at least the lower portion of the optic radiation.

CASE 5.—B. O., a man aged 40, gave a history that nine weeks before admission to the hospital he felt a shock "like electricity" running through his left lower limb. Associated with this he experienced dizziness and headache on the right. Three weeks later the left upper and lower extremities felt "asleep" and weak. The appetite became poor, and he lost weight.

Examination showed diminished sensation in the left extremities, which was of the cortical sensory type. There was slight paresis of the supranuclear part of the facial nerve on the left. The abdominal reflexes were absent, while the deep reflexes were increased on the left. The optic disks were slightly blurred. Central visual acuity was 10/15 in the right eye and 10/30 in the left. By perimetric examination a total crescentic depression in the left temporal field

of vision was found. On the following day the examination was repeated and the crescentic defect found. Systemic examination gave negative results.

Exploratory craniotomy over the right parietal lobe revealed no evidence of a neoplasm. The patient died on the tenth postoperative day. Autopsy revealed one metastatic carcinoma nodule located in the right parietal lobe. The tumor, 2.5 by 3 by 3 cm. in size, was 0.5 cm. below the lateral and mesial borders of the right parietal lobe; it extended from a point 0.5 cm. in front of the right parieto-occipital fissure anteriorly to a point 1 cm. in front of the splenium of the corpus callosum. The tumor was clearly demarcated from the rest of the brain. There were a few small hemorrhagic areas in the periphery, while on the lower edge there was some softening.

Comment.—This scotoma is another example of crescentic defect in the temporal field. The major lesion in this instance bordered the upper rim of the radiation. Detailed histologic studies of the optic fibers and the occipital cortex were not available.

CASE 6.—A. F., a man aged 49, entered the hospital with the history that one month prior to admission he saw spots before the eyes. One week later he had stiffness of the muscles of the neck, and while driving an automobile he felt "shaky" because he could not judge distances between his and other automobiles. For two weeks prior to admission he noted progressive failure in vision, the sensation being "as though there were a cloud over the eyes." He walked unsteadily, and on many occasions he bumped into objects and missed his way. He feared that he might fall through the floor because he was not sure of his gait. There was clumsiness of finer movements of the hands. He complained of headache in the frontal region and sticking pains in both eyes, of dizziness and of noises like "wheel grinding" in the ears. His relatives noted that his memory became impaired. He became mentally sluggish and emotionally unstable.

Examination revealed a palpable mass of glands in the left supraclavicular fossa, slight clumsiness of the left hand and increased reflexes on the left side of the body. Occasionally he made errors in the position sense of the left fingers and toes, and there was some dystereognosis in the left hand.

The fundi on admission revealed early papilledema; later this became advanced, and exudates and hemorrhages appeared. A few hemorrhages were noted to be present near the maculae. Mentally the patient was confused, dull and poorly oriented and had a defect in regard to memory and general information. A biopsy of the cervical lymphatic glands in the left supraclavicular fossa showed that the enlargement was due to follicular lymphoblastoma. The patient was not sufficiently mentally alert for adequate studies of his fields of vision to be made until thirteen days after admission, on April 3, 1936. At that time he was cooperative, and the studies revealed an unpaired total crescentic defect in the most peripheral portion of the left temporal field of vision (fig. 7A). The peripheral nasal field of the right eye was studied carefully by asking the patient to focus on a point 30 degrees along the horizontal meridian located to the right (temporally) of the central point of fixation, the absolute field of vision thus being exposed.

Perimetric tests for color showed a contracted temporal field in the left eye and a generally contracted field in the right eye. Central visual acuity was 20/100 in the right eye and 20/40 in the left. Examination of the central fields

by the stereocampimeter showed an enlarged blindspot and a relative scotoma in the temporal half of the central field of vision of the left eye.

Eight days later a ventriculogram demonstrated a mass in the right hemisphere. At operation on the same day a highly malignant type of sarcoma the size of a plum was removed from the posterior part of the right parietal lobe and the anterior part of the right occipital lobe. The tumor, which was located deep in the brain substance, was encapsulated and contained a loculated cyst.

Four days after craniotomy there was complete homonymous hemianopia in the left field. This soon began to recede, and after two weeks, on May 2, a large zone of central vision returned (fig. 7 B).

Comment.—This case illustrates that the crescentic defect found in the peripheral temporal field of vision of one eye could not be detected in the corresponding nasal field of the other eye even after the absolute field was studied. The factor of obstruction of the field by the body of the nose, which bounds the relative field of vision, was eliminated by having the patient rotate the eye being tested temporally in the horizontal meridian. It was interesting to note that when the hemianopia began to recede central vision was the first to reappear.

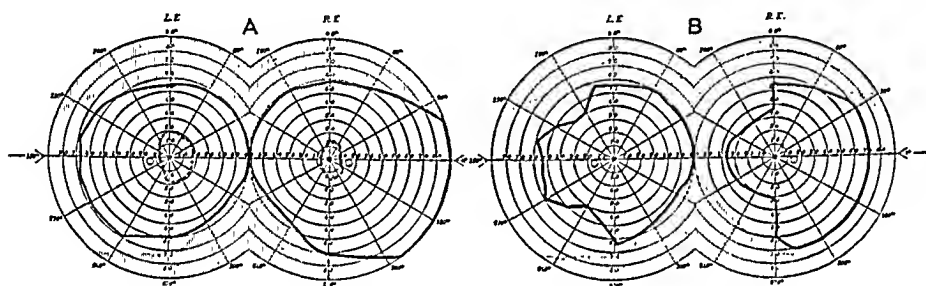


Fig. 7 (case 6).—There were crude visual hallucinations, inability to gage distances and cloudy vision. Central visual acuity was 20/100 in the right eye and 20/40 in the left. The fundus of each eye showed papilledema, with exudates and hemorrhages. There was a total crescentic defect in the left peripheral temporal field of vision (A). After the removal of the neoplasm from the right parieto-occipital lobe, there was complete homonymous hemianopia in the left field, which later regressed from the center toward the periphery (B). Examination was made with a test object 4 mm. in size.

PERIPHERAL TEMPORAL ABSOLUTE SCOTOMA AND RELATIVE CRESCENTIC SCOTOMA

CASE 7.—F. C., a 52 year old woman, was admitted to the hospital complaining of headache of three weeks' duration. Two and a half weeks before admission she forgot how to write and had attacks of dizziness and weakness. During the week before admission vomiting developed; she became somewhat confused and had slight difficulty in understanding spoken and written language.

Examination revealed a slight increase in the muscular tone and in the reflexes on the right side of the body. There were minimal paresis in the right upper limb, mild hemihypalgesia on the right and bilateral papilledema with exudates

and hemorrhages. On gross examination a slight defect was seen in the right temporal field of vision. The patient had total alexia and agraphia. Spontaneous speech was hesitant and showed several minor errors. She was unable to concentrate. She appeared bewildered and depressed. The spinal fluid was slightly xanthochromic and under a pressure of 160 mm. of water; it contained 69 lymphocytes per cubic millimeter, and its total protein content was 84 mg. per hundred cubic centimeters of fluid.

On the day after the patient entered the hospital, on Aug. 19, 1934, perimetric examination revealed a crescent of absolute blindness in the right peripheral temporal field of vision and, mesially adjacent, a crescent of indistinct or relative blindness (fig. 8*A*). Because the patient became fatigued easily, the fields for color were not tested. Central visual acuity was 20/20 in the right eye and 20/50 in the left. Two days later the perimetric tests showed a slightly larger absolute

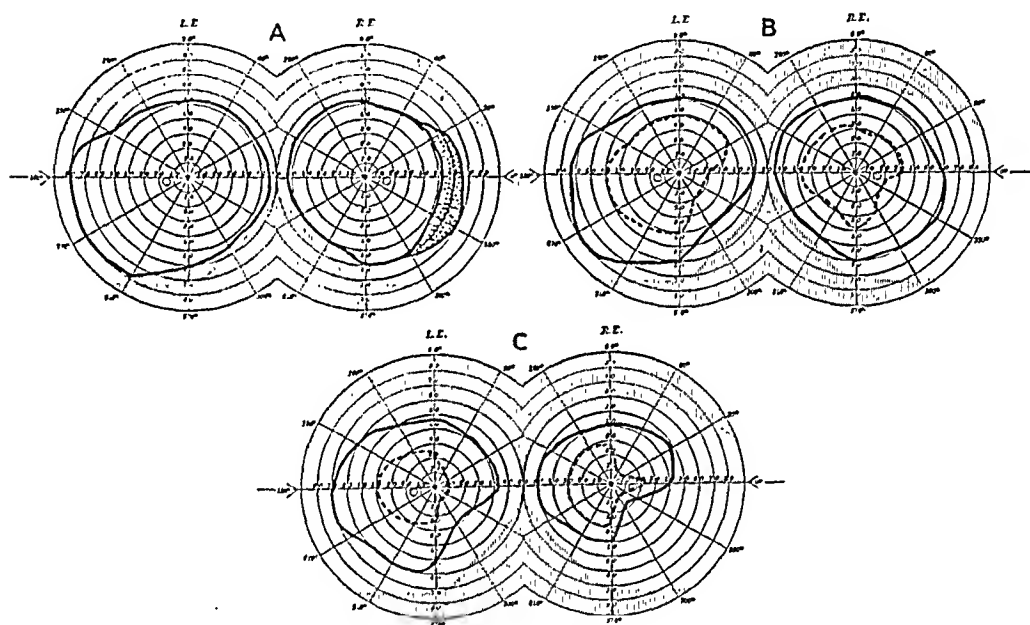


Fig. 8 (case 7).—There were alexia and agraphia. Central visual acuity was 20/20 in the right eye and 20/50 in the left. The fundus of each eye showed papilledema with hemorrhages. There were a total absolute and a relative crescentic scotoma in the right peripheral temporal field of vision, as shown in *A*. (The stippled area represents the crescent of indistinct vision.) Note that the progression of the hemianopia from the periphery toward the center was preceded by defects in the field for color, as shown in *B* and *C*. Examination was made with a test object 4 mm. in size.

crescentic defect, a small contraction in the left inferior peripheral nasal field and homonymous inferior incomplete quadrantanopia in the right field for color (fig. 8*B*).

On August 25, seven days after admission to the hospital, a ventriculogram taken after trephining in the right occipital region demonstrated a mass in the region of the left posterior horn. On the day after the studies with air were made, perimetric examination showed incomplete homonymous hemianopia in the right field, with a tendency for the inferior quadrants to be more defective (fig. 8*C*). Associated with this there was a homonymous hemiachromatopia in the right field, in which central vision was preserved.

On the following morning craniotomy revealed an infiltrating glioma (spongioblastoma polare) in the posterior and inferior portions of the left parietal lobe, located 5 cm. below the surface of the brain.

Comment.—In this instance there was a crescentic defect which was split in the longitudinal meridian, causing an absolute peripheral crescent and, mesial to this, a crescent of transient or "relative" blindness. Equally interesting was the progression of the unpaired crescentic defect to homonymous incomplete hemianopia and quadrantanopia. It should be noted that the defects in the field for color preceded those in the field for form, indicating that more "central fibers" were implicated, as well as the "peripheral fibers," and that the hemianopia had advanced from the periphery.

CASE 8.—E. G., a 52 year old man, entered the hospital complaining of difficulties in memory and in calculation. Three years before admission he sustained an injury to the head, at which time amber-colored fluid escaped from the left ear.

Five months prior to admission he experienced numbness of the fingers in the right hand. Three months later he had a fainting spell, and since then he had felt "pressure" in the back of the head. In addition he complained of impairment of memory, had difficulty in recalling names of persons and numbers and made mistakes in calculations and in reading printed words. On occasions he jumbled his words in conversation.

Examination revealed normal spontaneous speech. However, careful analysis showed that the patient omitted and transposed letters in writing, mixed syllables in reading and made errors in calculation, especially in subtraction and division. Previously he had been an excellent mathematician. He also had echolalia. Lumbar puncture showed a clear fluid under a pressure of 210 mm. of water. A roentgenogram of the skull revealed a calcified pineal body in the normal position.

On Nov. 23, 1934, two days after admission, perimetric tests disclosed a total crescentic defect in the right peripheral temporal field of vision (fig. 9 A).

The field for color in the right eye showed a tendency to incomplete homonymous inferior quadrantanopia. Reexaminations on the following day and on two subsequent occasions showed an absolute crescentic defect in the field for form and that for color in the right peripheral temporal field of vision; mesial to this defect was a crescentic relative scotoma. The fields in the left eye were normal (fig. 9 B). Central visual acuity, uncorrected, was 10/50 in the right eye and 10/20 in the left.

The fundi were myopic and showed a slight myopic conus in each eye. Nine days after admission an encephalogram indicated that a mass was present in the region of the left temporal lobe. At exploration a tumor the size of a hickory nut was removed from the posterior portion of the junction of the left temporal and parietal lobes; it was at a depth of 5 cm. Autopsy later showed the cavity which was left by the removed tumor to be located deep in the midportion of the left temporal lobe, lateral to the convexity of the inferior temporal horn of the lateral ventricle.

Comment.—This is an example in which two crescents were demonstrable in one peripheral temporal field of vision, the more peripheral scotoma being absolute and the more central being relative. Equally interesting were the findings in the fields for color, which also showed the temporal crescent defect in the right eye. Also of note was the slight incipient defect in the field for form found in the inferior nasal quadrant of the left eye. The homonymous inferior quadrantanopia in the right field for color seemed to be confirmatory evidence for the foregoing defects in the visual field. The quadrantic defects were not found on subsequent examinations. The recession of the defect in the field may have been due to fluctuations in the course of the disease, which are often observed in cases of tumor of the brain.

CASE 9.—A. P., a 27 year old art student, entered the hospital with the history that two years before admission she imagined she saw flashes of light. Two weeks before admission she vomited and suffered headache in the frontal region and dizziness. A day before admission there were double vision and numbness in the right arm.

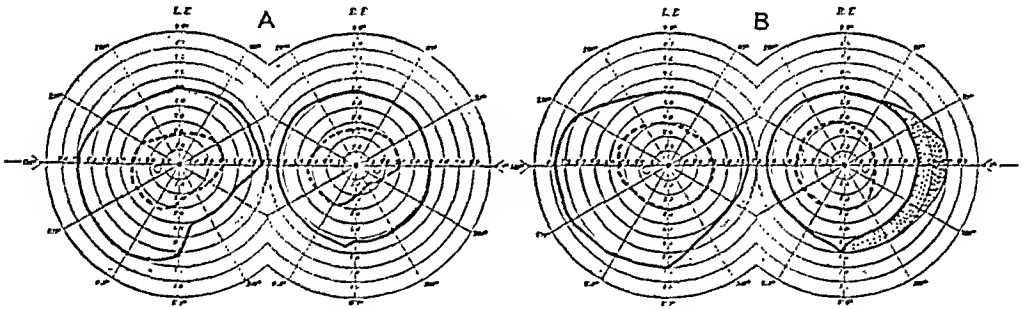


Fig. 9 (case 8).—There were dyslexia and dysgraphia. Central visual acuity was 10/50 in the right eye and 10/20 in the left. The fundus of each eye showed myopia. The fields (A) were similar to those in figure 8 B. Two days later, on November 25, the defect regressed, and the crescent of indistinct vision (the stippled area) appeared (B) as in figure 8 A. Examination was made with a test object 4 mm. in size.

On examination the positive findings were: hemihypalgesia and facial weakness on the right, blurring of the margins of the optic disks, mental dulness, inability to concentrate and euphoria. The fields of vision were not plotted; grossly there was no hemianopia. A roentgenogram of the skull showed increased convolutional markings and a calcified pineal gland which was displaced to the right. A ventriculogram followed by craniotomy revealed a glioma the size of a walnut in the left parietal lobe. The patient made an uneventful recovery.

Five months later she was readmitted to the hospital because of headache and vomiting. At this examination there were dystereognosis and impaired position sense in the right extremities. There was slight aphasia, with dyslexia and dysgraphia. The fundi were normal. The cranium was reexplored, and more tumor tissue was removed from the same region.

One year after the first operation the patient was readmitted a third time because of the same symptoms. It was reported that she had a visual hallucina-

tion, the details of which could not be ascertained. Physical examination showed more marked sensory changes and aphasia than on previous admissions. She was extremely cooperative in all kinds of performance tests. Gestalt tests showed her to be a "visual" subject. Although she had slight difficulty in reading and writing, she was able to draw objects at sight with great skill and in detail. Plotted visual fields showed an absolute crescentic scotoma and mesial to this a relative crescentic scotoma, in the right peripheral temporal field of vision. There was homonymous hemianopia for color in the right field, which was more marked for green than for red. Central visual acuity was 20/40 in the right eye and 20/30 in the left. The fundi were normal. The patient was operated on a third time, and at this operation the glioma was found at the juncture of the left parietal, temporal and occipital lobes. Six weeks after the last operation complete homonymous hemianopia was found by confrontation tests.

Comment.—Despite the fact that the visual fields were plotted some time after the original operation, this case may be used as another example of absolute peripheral temporal crescentic scotoma and relative crescentic scotoma. The incomplete homonymous hemianopia for color,

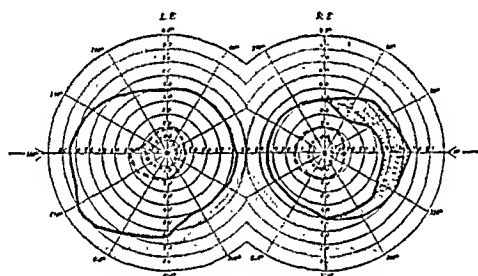


Fig. 10 (case 9).—There were crude and complex visual hallucinations, with dyslexia and dysgraphia. Central visual acuity was 20/40 in the right eye and 20/30 in the left. The fundus of each eye showed slight blurring of the margins of the optic disk. There were a total crescentic scotoma in the right peripheral temporal field of vision and, mesial to this, a crescentic relative scotoma (the stippled area). There was incomplete homonymous hemianopia for color, which was greater for green (the field bordered by the dash line) than for red (the field bordered by the dotted line).

which was more marked for green than for red, indicated that function of many fibers in the dorsal and ventral halves of the optic radiation had been impaired.

CASE 10.—J. W., a 42 year old man, was admitted to the hospital with the history that two weeks prior to admission he was found on the bathroom floor in an unconscious and rigid state. A few days later there developed stiffness of the neck and inability to read or to find words or to concentrate properly. One day before admission he had headache and nausea.

Examination revealed a pulse rate of 64 beats per minute. There was mild sensory aphasia, with dyslexia, dysgraphia and dysnomia. Paresis of the right-central portion of the face and slight signs of involvement of the pyramidal tract on the right were evident. Mentally, the patient was irritable; he appeared

depressed and somewhat bewildered. His memory for immediate events was markedly impaired. The fundi showed slight blurring of the nasal margins, and the veins appeared engorged. Perimetric tests revealed homonymous hemianopia for color, with preservation of central vision, an irregularly crescentic area of absolute blindness in the right peripheral temporal field of vision and, centrally adjacent to this, an irregularly crescentic zone of partial blindness (fig. 11). There was a tendency for the defects to be greatest in the inferior halves of the crescents. In the other eye, along with a slight defect in the inferior nasal quadrant, there appeared to be incomplete incongruent homonymous inferior quadrantanopia. Central visual acuity was 20/20 in each eye.

Four days after admission exploratory craniotomy was performed. The patient died one month later. At autopsy a tumor (spongioblastoma multiforme) was observed in the ventral half of the temporal lobe, involving the posterior portions of the optic radiation. Surrounding this area there was edema.

Comment.—This case demonstrates the relative crescentic defect, and since such a zone of indistinct vision has been found in three previous cases, it begins to assume significance. It would appear that the

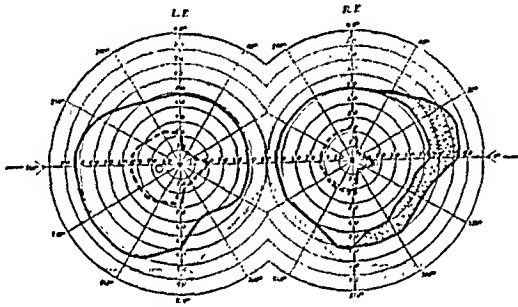


Fig. 11 (case 10).—There were dyslexia and dysgraphia. Central visual acuity was 20/20 in each eye. The fundus of each eye showed slight blurring of the nasal margins of the disk. On examination with a test object 4 mm. in size a relative crescentic scotoma (the stippled area) in the right peripheral temporal field of vision and incipient homonymous hemianopia for color in the right field, with preservation of central vision, were found.

temporal crescent may be split longitudinally, and this suggests the possibility that the fibers in the radiations have a lamellar distribution and are arranged in definite order. The incongruity of the hemianopia in the field for color and the disproportion between the field for form and that for color indicated an active process in which the optic fibers were irregularly affected.

GENERAL COMMENT

In the foregoing cases of neoplasm implicating the optic radiations it has been shown that crescentic or hemicrescentic areas of blindness may be found in the peripheral temporal field of vision of one eye. In corroboration of this observation were the concomitant defects found in the field for color (cases 4, 6 and 8) and the crescentic relative scotoma

which were found to be mesially contiguous to the peripheral temporal crescentic absolute scotoma (cases 7, 8, 9 and 10). Cases illustrating a sparing of hemicrescentic and crescentic areas in the peripheral zone of the temporal half of the field with homonymous hemianopia have been described by Harris,¹⁰ Behr,¹¹ Delepine¹² and Traquair.⁵

These clinical observations, as well as anatomic studies of experimental and pathologic specimens, gave ample proof that the most peripheral portions of the temporal field of vision are unpaired and unilaterally represented in the opposite optic cortex. The exact route of the fibers from the retina to the lateral geniculate body was traced by Brouwer and Zeeman,¹ and that from the lateral geniculate ganglion to the calcarine cortex by Poliak.³ Prior to 1932 the location and distribution of the optic fibers in the radiation were moot points. Pfeiffer¹³ concluded that the upper rim of the lateral portion of the optic radiations contained the fibers for central vision and that the lower rim contained the fibers for peripheral vision. From this one may infer that the fibers for vision in the temporal crescent traverse the ventral part of the optic radiation.

Duke-Elder¹⁴ stated that these fibers have a special localization in the central optic pathways, in the anterior part of the medullary optic lamina, being finally projected in the anterior part of the optic cortex. Some authors are of the opinion that the fibers are in the mesial portion of the ventral rim. From our clinical studies it appears that the fibers for peripheral temporal vision may be contiguous with their respectively paired quadrants of paracentral and central vision, the arrangement being lamellar (fig. 1). It is probable that the fibers for monocular vision (in the temporal crescent) do not intermingle with those subserving binocular vision.

The optic radiation is composed of individual bundles, each having its definite subcortical origin, its course and its cortical termination. Our observations are in agreement with those made in the anatomic studies of Poliak.³ He summarized that the most dorsal bundles of the optic radiation transmit vision from the opposite upper peripheral

10. Harris, W.: Hemianopia with Especial Reference to Its Transient Varieties, *Brain* **20**:308, 1897.

11. Behr, C.: Die homonymen Hemianopsien mit einsietigem Gesichtsfelddefekt, im "rein temporalen halbmondförmigen Bezirk des binokularen Gesichtsfeldes," *Klin. Monatsbl. f. Augenh.* **56**:161, 1916.

12. Delepine, S.: Case of Right Lateral Homonymous Hemianopsia: Arteriosclerotic Softening of the Left Cuneus, *Tr. Path. Soc. London*, **41**:2016, 1890.

13. Pfeiffer, R. A., in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 1, p. 426.

14. Duke-Elder, S.: *Text Book of Ophthalmology*, St. Louis, C. V. Mosby, 1934, vol. 1, p. 273.

nasal retina, which in the field of vision corresponds to the lower peripheral temporal hemicrescent, while the most ventral bundles transmit vision from the contralateral lower peripheral nasal retina, which receives impulses from the upper peripheral temporal hemicrescent in the field of vision (fig. 1).

DIRECTION IN WHICH HOMONYMOUS ANOPIAS DEVELOP

Irrespective of the location of the lesion in the optic radiation, homonymous lateral anopias have been observed to begin in the periphery with a crescentic or hemicrescentic scotoma in the temporal field of vision (cases 2, 3, 7 and 9). Subsequently the blindness progressed toward the center and frequently did not involve central vision. During the process of regression the reverse phenomena occurred (cases 6 and 8)—the range of central vision gradually increased to include the paracentral, and finally the peripheral, field of vision. The foregoing statement is illustrated by the following case report:

CASE 11.—A 34 year old school teacher was admitted in stupor to a neighboring hospital. Examination showed a stiff neck, fever, glycosuria and 30 lymphocytes per cubic millimeter of spinal fluid. Three days later she was transferred to the Mount Sinai Hospital, and on admission mixed aphasia and weakness of the right-central part of the face were found. Under observation, defects in the right field of vision appeared and disappeared, as shown in the charts (fig. 12).

The diagnosis rested between neoplasm of the left temporal lobe and encephalitis. An encephalogram showed no abnormality. The patient was discharged from the hospital in a much improved condition. The nature and location of the lesion were not verified.

This patient showed the temporal crescentic defect at the onset and end of homonymous hemianopia.

Although Peter,¹⁵ in his book on perimetry, stated that anopias may begin in the center of the field and develop peripherally, such observations have not frequently been made in cases of tumors of the temporal lobe. Globus and Silverstone¹⁶ noted that there was no essential difference in the type of defects of the visual field in cases of tumor which involved the mesial or lateral portions of the temporal lobe. In most instances of homonymous lateral anopias due to lesions of the radiation the defect does not include central vision. If the fibers for central, paracentral and peripheral vision have a definite position in the suprageniculat pathways, why should not a lesion destroying, for example, the fibers for central or paracentral vision produce

15. Peter, L. C.: *The Principles and Practice of Perimetry*, Philadelphia, Lea & Febiger, 1931.

16. Globus, J. H., and Silverstone, S. M.: *Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances*, Arch. Ophth. **14**:325 (Sept.) 1935.

homonymous anopia starting in the central or paracentral zones and developing toward the periphery rather than starting from the periphery and developing toward the center? Why is it that central vision is the last to disappear and the first to recover, irrespective of which fibers of the optic radiation are destroyed first?

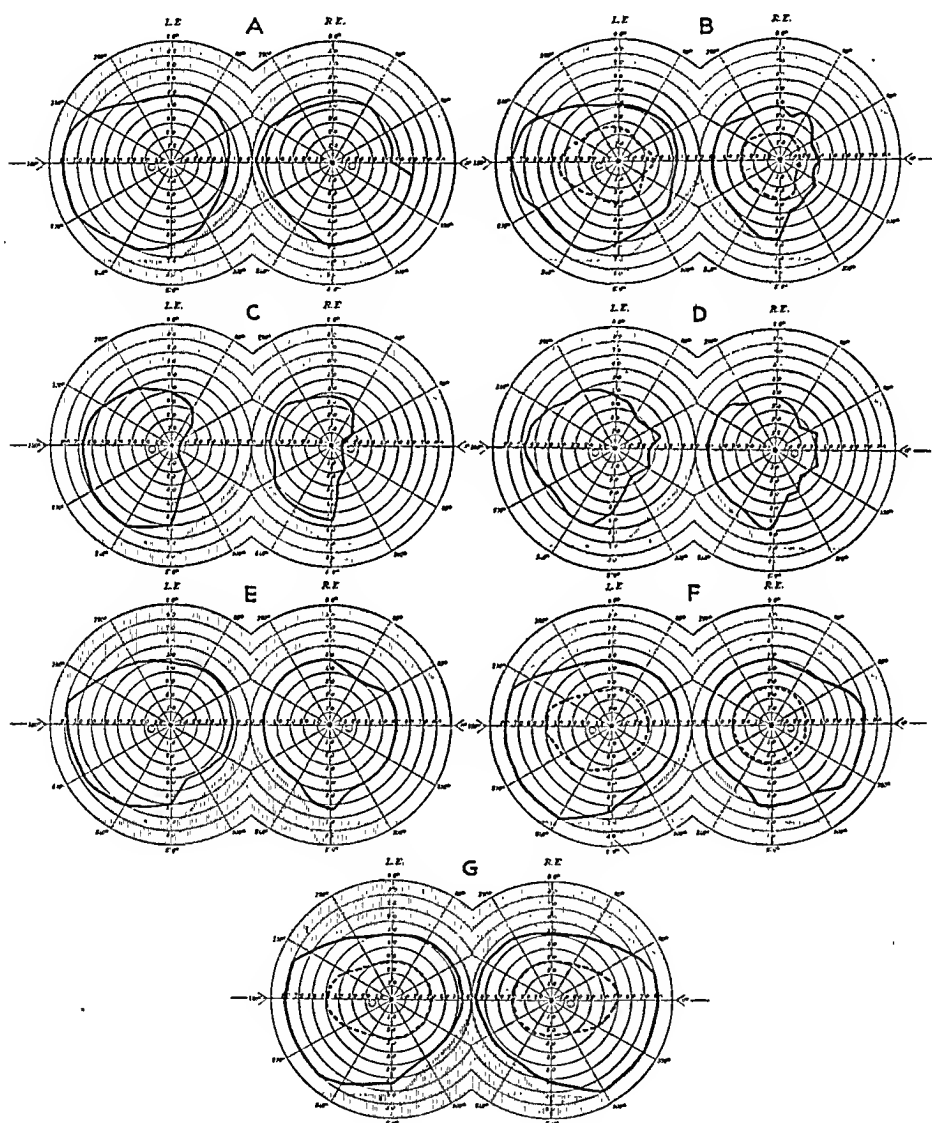


Fig. 12 (case 11).—There were alexia and agraphia. Central visual acuity was 20/20 in each eye. The fundi were normal. The charts of the visual fields show the development and regression of homonymous hemianopia in the right field. The letters indicate the fields on the following dates: *A*, Jan. 4, 1934; *B*, January 7; *C*, January 9; *D*, January 13; *E*, January 26; *F*, February 20, and *G*, March 1. Note that the peripheral temporal crescent in the field for form and in that for color was the first to be affected and the last to return to normal. Examination was made with a test object 4 mm. in size.

The answer to these questions may be derived from the following observations and theories. The macular and paracentral fields constitute the binocular field of vision. The periphery of the temporal field of vision is monocular (not paired in the homonymous nasal field) and unilaterally represented. Since the eyes operate as a unit, it is probable that structures subserving vision for the paired central and paracentral fields and the unpaired peripheral temporal fields are closely related in their anatomic and physiologic features.

Traquair⁵ has shown that sensitivity to a white object, or visual acuity in the relative field of vision, was highest in the center and gradually decreased toward the periphery. Visual acuity varies with the density of the retinal elements (rods and cones). The number of rods and cones per unit area is greatest in the central region and least in the periphery. Brouwer and Zeeman¹ traced the optic fibers from the retina and found a progressive increase in the area occupied by the fibers for central vision as they approached the lateral geniculate body. The unpaired peripheral fibers occupied a narrow rim in the ventral portion of the external geniculate ganglion (fig. 1). Pfeiffer¹³ traced the fibers farther along the geniculocalcarine pathway through the external sagittal stratum of the optic radiation. He stated that in the dorsal part of the stratum sagittale externum coursed the macular fibers. Henschen¹⁷ concluded that the bundle of fibers for peripheral vision occupied a part which was 1 cm. high in the ventral portion of the external rim; the greatest area was occupied by the fibers for macular vision. Poliak³ inferred that about half of the area of the entire optic radiation represents central vision. Holmes,¹⁸ from his studies of gunshot wounds in the occipital lobe, proposed that the serial concentric zones in the retina from the macula outward to the periphery seem to be represented in the same order from behind forward in the occipital cortex, and that the macular area is relatively large, while the peripheral area is relatively small. The relative sizes of the areas covered by the fibers for central, paracentral, and peripheral vision are illustrated in figure 1.

Thus it is seen that central vision is transmitted by fibers greater in number than those which represent the paracentral or the peripheral field of vision. The area occupied by fibers for macular vision in the geniculocalcarine pathways gradually increases as they approach the occipital cortex. It is probable that the change in the number of optic fibers and in the area covered by these in the retinogeniculocalcarine

17. Henschen, S. E., quoted by Pfeiffer,¹³ p. 436.

18. Holmes, G.: Disturbances of Vision by Cerebral Lesions, *Brit. J. Ophth.* 2:353, 1918.

pathway is proportional to the gradual change in the visual acuity at their corresponding points in the field of vision. In other words, a direct relationship exists between function and structure.

Physiologically, an interesting and important feature in the intensity of the defects is the relation of the loss in the field for color to that in the field for white (cases 1, 4, 6, 7, 8, 9 and 10). In partial impairment of function, vision for color is first diminished. Only when the impairment has reached a certain degree is vision for white demonstrably reduced. In a diseased area either a large number of nerve fibers may be slightly affected, the remainder being healthy or seriously damaged, or, on the other hand, there may be a small percentage of severely damaged nerve elements fairly evenly distributed, all the others being healthy or a few being slightly diseased. In both instances visual acuity for white and for color is reduced, but in the first instance the loss of vision for color is excessive. The relative numbers and the distribution of the healthy, partly damaged and severely damaged nerve structures vary according to the nature and location of the lesion. The nature of the intensity of the corresponding defect in the field may safely be regarded always as the resultant of the aforementioned factors. The relation between the uniformity of the defect and that of the lesion is complicated by the varying value of the visual acuity between the center and periphery of the field and by the question whether by the same degree of damage functional impairment is as easily produced in one set of fibers as in another.

With these theories in mind it should become clear why a lesion, especially a tumor, located anywhere near or in the optic radiation will be manifested by homonymous anopia which begins in the periphery and gradually develops toward the center. Because the fibers for peripheral vision are fewest, cover the least area and, proportionally, conduct vision of lowest acuity, a tumor that directly or indirectly destroys the function of a few of these fibers will promptly reduce the visual acuity in the corresponding field. On the other hand, the tumor may affect primarily or simultaneously a greater portion of the fibers for central or paracentral vision and yet, because the optic fibers occupy a larger area in the radiation, are greater in number and conduct vision of increasing acuity, the visual acuity in the corresponding field will decrease in the periphery first, for the reasons already given. When the lesion affects the fibers of either the dorsal or the ventral half of the radiation, the defect in the visual field will start as a hemicrescentic scotoma in the periphery of the temporal field and develop into homonymous quadrantic anopia (case 2); if the lesion progresses to involve the other half of the radiation (as in case 3—the dorsal half

after the ventral half), homonymous anopia will occur, and if the edema or the tumor affects one half of the radiation more than the other (as in case 7), irregular quadrantic defects in the field for form and hemianopia in the field for color will be found. When the disease process affects the central fibers of both halves of the geniculocalcarine pathway, there will follow homonymous anopia, at first for color (case 10) and then for form (case 11).

The same explanation applies to regressing hemianopia in which the pathologico-physiologic condition is reversed. Although these deductions may explain preservation of central vision in hemianopia caused by a lesion implicating the optic radiation, they do not confirm or disprove that macular vision may be bilaterally represented.

Objections to the foregoing theories may be advanced from examples of hemianopia in which peripheral temporal vision was spared. In most of those cases the lesions were unverified and the localization was based on clinical grounds. In the cases in which the disease process was verified (usually cases of arteriosclerosis or gunshot wounds), the location was in the cortex of the occipital lobe, where the physiologic and structural components for vision are different from those of the optic radiations in the temporal lobe.

Riddoch¹⁹ studied recovery from hemianopia following gunshot wounds, and he found that recovery occurred from the periphery to the center of the field. The remaining defect in the central or paracentral field was loss of appreciation of movement; this zone was surrounded by a zone of loss of recognition of objects. In some instances he found regression beginning with a temporal crescent. These observations are in direct contrast to those made in patients with tumor of the brain and require explanation. The location of the lesions in Riddoch's cases were estimated from the points of entrance and exit of the missile and, in some cases, by operation. The extent of the lesion could not be adequately determined. In some of Riddoch's patients the defects discovered seemed to be migrating from one field to another, implying that they were progressive or regressive bilateral lesions of the optic pathways. All the lesions that he found were in the occipital lobe. Furthermore, it must be remembered that in cases of gunshot wounds the damage to the tissues in question is sudden and massive. The lesion is at its maximum in the first few days. When the surrounding edema disappears there is a residual defect in the optic pathways or the occipital cortex. Thus, at first there will be complete homonymous hemianopia, which later decreases to a focal scotoma, the

19. Riddoch, G.: Dissociation of Visual Perception Due to Occipital Injuries, with Especial Reference to Appreciation of Movement, *Brain* 40:15, 1917.

nature of the latter depending on the number of fibers or cells destroyed and the location of the scar in the geniculocalcarine pathway. In cases of tumor of the brain the sequence of events is different. The disease process is expanding and usually continual. The anopias in cases of tumor of the temporal or parietal lobe are produced more likely by reactive phenomena than by direct neoplastic infiltration of the broad but very thin band of optic fibers which constitute the optic radiation. The recovery in the field of vision which is found at some interval after extirpation of the tumor is due to decrease of pressure, edema or other effects which impeded the function of the optic fibers. Discrete residual scotoma may be observed if a part of the band of radiation is destroyed during the operation. Unfortunately, most neoplasms which involve the radiation directly and produce cerebral symptoms are large, so that in removal of the tumor most of the radiation may be destroyed, and there will be no recovery of visual function. Even if only a part of the optic fibers are extirpated with the neoplasm, by the time the partially damaged fibers would have recovered function (and a great number of fibers would be impaired if the tumor were in that location), the neoplasm would probably recur. Another reason why residual scotomas are not commonly recorded in cases of tumor of the brain postoperatively is that even if the patient lived long enough he might not be available for observation.

Thus we saw that the earliest sign of involvement of the optic radiation was a crescentic defect in the peripheral portion of the opposite temporal field of vision. Strauss²⁰ emphasized the necessity for studying cases of tumor of the brain early in the course of the disease. Such a practice will undoubtedly permit the temporal crescentic scotoma to be more commonly recognized than it has been. Indeed, this defect may even be found to be the most frequent type of distortion in the fields of vision of patients with tumor of the brain.

SUMMARY

Cases are reported in which a lesion of the optic radiation produced an unpaired crescentic or hemicrescentic defect in the periphery of the temporal field of vision. In several instances there was a concomitant defect in the field for color.

In four cases an unpaired crescentic relative scotoma was found mesial to the peripheral crescentic absolute scotoma. This observation suggested the probability that the fibers in the optic radiations have a lamellar arrangement.

20. Strauss, I.: The Initial Symptoms and Early Diagnosis of Tumor of the Brain, *Bull. New York Acad. Med.* **12:467** (Aug.) 1936.

The most dorsal bundles of the optic radiation transmit vision from the opposite upper peripheral nasal retina, while the most ventral bundles transmit vision from the contralateral lower peripheral nasal retina.

Irrespective of the location of the lesion in the radiation, homonymous anopias usually begin in the periphery and advance toward the center, frequently sparing central vision. The regression of the anopias occurs in the reverse manner. These phenomena are explained on a functional and structural basis.

An unpaired peripheral temporal scotoma indicates an early defect in the optic radiation. Such a scotoma has localizing value in the early diagnosis of tumor of the brain.

ASTIGMATIC DIALS IN REFINED REFRACTION

JAMES J. REGAN, M.D.

BOSTON

I believe, as John Green¹ stated in 1866, that astigmatic dials, properly used, are the most dependable practical devices yet discovered for measuring the amount and axis of astigmatism. Failure is due to faults of equipment or technic. Lancaster² was too modest to say this in 1915 when he presented his paper on subjective tests for astigmatism, but if one had read between the lines or taken the trouble to test himself or his patients by this method, one would appreciate why this master of the art of refined refraction anticipated a rational response, namely, the general adoption of his dial or other astigmatic dials by ophthalmologists.

Twenty years later one finds earnest, conscientious, painstaking refractionists struggling without the satisfaction which astigmatic dials would afford or, perhaps, using only one dial—the clock-face or sunburst dial—with disappointing results. What is the reason? The chief reason is that most textbooks containing chapters on refraction and even textbooks on refraction deal with the technic for the use of astigmatic dials in so inadequate a fashion that beginners, after trying to follow the incomplete directions, are not satisfied with the results and give up this method to seek some more reliable method for determining the presence, amount and axis of astigmatism—and, incidentally, there is none.

Few American authors and fewer European authors have written about the use of astigmatic dials. Directions have been brief and have usually dealt with the use of the clock-face dial, which, if used without a supplementary dial, is inadequate, for the following reasons:

1. The lines 30 degrees apart do not admit fine discrimination of axes.

2. If the examiner attempts to neutralize the astigmatism (to determine the amount of astigmatism), by using one dial, he finds that before the lines in the principal meridians (for example, the vertical and horizontal lines) become equally black, the patient states that the oblique lines and not the vertical (or horizontal) lines appear blackest.

1. Green, John: Annual Report of the Netherlands Ophthalmic Hospital, Utrecht, 1866.

2. Lancaster, W. B.: Subjective Tests for Astigmatism, Especially Astigmatic Charts, Tr. Am. Acad. Ophth., 1915, p. 167.

It appears, therefore, that a supplementary dial of two principal meridians, as suggested by Verhoeff³ in 1899 must be used in conjunction with the clock-face (or sunburst) dial to obtain the best results. My experience with Green's, Verhoeff's, Thorington's and Friedenwald's dial and with other dials has resulted in the conclusion that Lancaster's 50 cm. dial with lines 20 cm. long, about 2 minutes wide and 10 degrees apart, is the most refined. Regarding a supplementary dial with which to determine the amount of astigmatism, I think that equal accuracy can be obtained with single crossed lines of different lengths and widths; but, as Lancaster pointed out, since the patient's vision must be fogged to obtain accurate results, the lines must be wider than 1 minute.

Green's¹ original lines, as well as Wallace's,⁴ were designed "to subtend an angle of one minute for the purpose of making recognition of the lines in any one meridian the standard of perfect vision in that meridian." After working with numerous dials between 1866 and 1878, Green concluded that the lines on some of his original dials were too narrow for the best results at six meters.⁵ He found that better results were obtained by using wider lines. This was confirmed by Lancaster, clinically and photographically. Nevertheless, many writers have failed to appreciate the importance of this point. Thus, Thorington⁶ stated that in most charts the lines subtend an angle much greater than the Snellen standard for the distance at which they are used, and "in this way the true delicacy of the test for small errors or amounts of astigmatism is sacrificed." Friedenwald's charts fall into the same error. Charts with narrow lines like Friedenwald's may give better results than Lancaster's for a small percentage of patients, viz., those few patients who combine unusually good powers of observation with small errors of refraction. It should be distinctly understood that what I am maintaining is that wider lines give better results for a larger percentage of patients.

The simplicity and economy of astigmatic dials should make them the universal choice of refractionists, especially those who, from choice or necessity, work without a cycloplegic. Helmholtz⁷ used small figures made up of numerous radiating lines to illustrate the general phenomena

3. Verhoeff, F. H.: Two New Astigmatic Charts, *Ophth. Rec.* 8:541, 1899.

4. Wallace, J.: An Improved Astigmatic Chart, *Univ. Med. Mag.* 2:13, 1889-1890.

5. Green, J.: On a New System of Tests for the Detection and Measurement of Astigmatism, *Tr. Am. Ophth. Soc.* 4-5:131, 1867-1868.

6. Thorington, J.: *Refraction and How to Refract*, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1904, p. 138.

7. Helmholtz, H. L. F.: *Handbuch der physiologischen Optik*, Leipzig, L. Voss, 1909, pl. 2, fig. 5.

of astigmatic vision, and Donders⁸ in 1860 or earlier used a star-shaped figure made up of fine radiating lines as a practical test for astigmatism. Snellen adopted a dial similar to Green's "B" dial, but reduced the number of lines from sixty (3 degrees apart) to eighteen (10 degrees apart) and used only the upper half—the astigmatic fan—regarding which Green⁹ had said that it "has the single advantage of falling within the limits of the Octavo page of his test types."

That the presence and axis of astigmatism can be demonstrated by dials is therefore an old story, but pitfalls due to lack of reliable technic must have been many, else, in my opinion, no refraction would be concluded today without resorting to the dials.

Evidence tending to prove the superiority of the Lancaster dials and his technic for obtaining the best clinical results with them were presented so lucidly and convincingly by Lancaster² in 1915 that one has but to read the article, procure the charts and proceed to better results in refraction. True, the original charts are not on the market and must be made by gluing black velvet ribbon on wedding stock paper, but Bausch and Lomb¹⁰ manufactured a set of greater durability. These were square boxes about 2 inches (5 cm.) in depth, with the lines cut out on metal fronts, but the cost of these seems to have been more than most refractionists wished to pay.

Another drawback to the popularity of the dials is their size. They require more wall space than many wish to give, or can give, to dials. Lancaster explained that if the dial is small, "a 10° interval brings the lines pretty close together"; in his larger dial his intention was to avoid this and at the same time have the lines 10 degrees apart, this arrangement being, I believe, superior to that in which the lines are 15 or 30 degrees apart.

The aforementioned difficulties, viz., the trouble (and skill) required to make the dials, the expense of the manufactured set and the economy of wall space, are all legitimate deterrents to the adoption of the dials, especially by the beginner, unless, of course, he has used them while in training and already considers them indispensable. When Lancaster's attention was called to this, he offered to aid me in my effort to modify his original dials. After months of trial we produced the dials here offered, which have given results almost as satisfactory as the original Lancaster dials. These dials are now available at nominal cost and require about the same wall space as Verhoeff's dials.

8. Donders, F. C.: Beiträge zur Kenntnis der Refraktions- und Accommodationsanomalien, *Arch. f. Ophth.* (pt. 1) 7:155, 1860.

9. Green, J.: Test-Diagrams for the Detection and Measurement of Astigmatism, *Tr. Am. Ophth. Soc.* 2:467, 1878.

10. Bausch and Lomb issued a booklet giving instructions by Lancaster in the use of the charts.

SUGGESTED EXPERIMENTS WITH LANCASTER-REGAN ASTIGMATIC DIALS TO SHOW THE PRINCIPLE INVOLVED

Those who are not skilled in the use of astigmatic dials (and this includes some who think they are but who are not) will find the following experiments a valuable means of clarifying the principles involved.

How to Illuminate the Dials.—The charts are hung in a good light of at least 15 to 25 foot-candles, the whole wall where they are hung

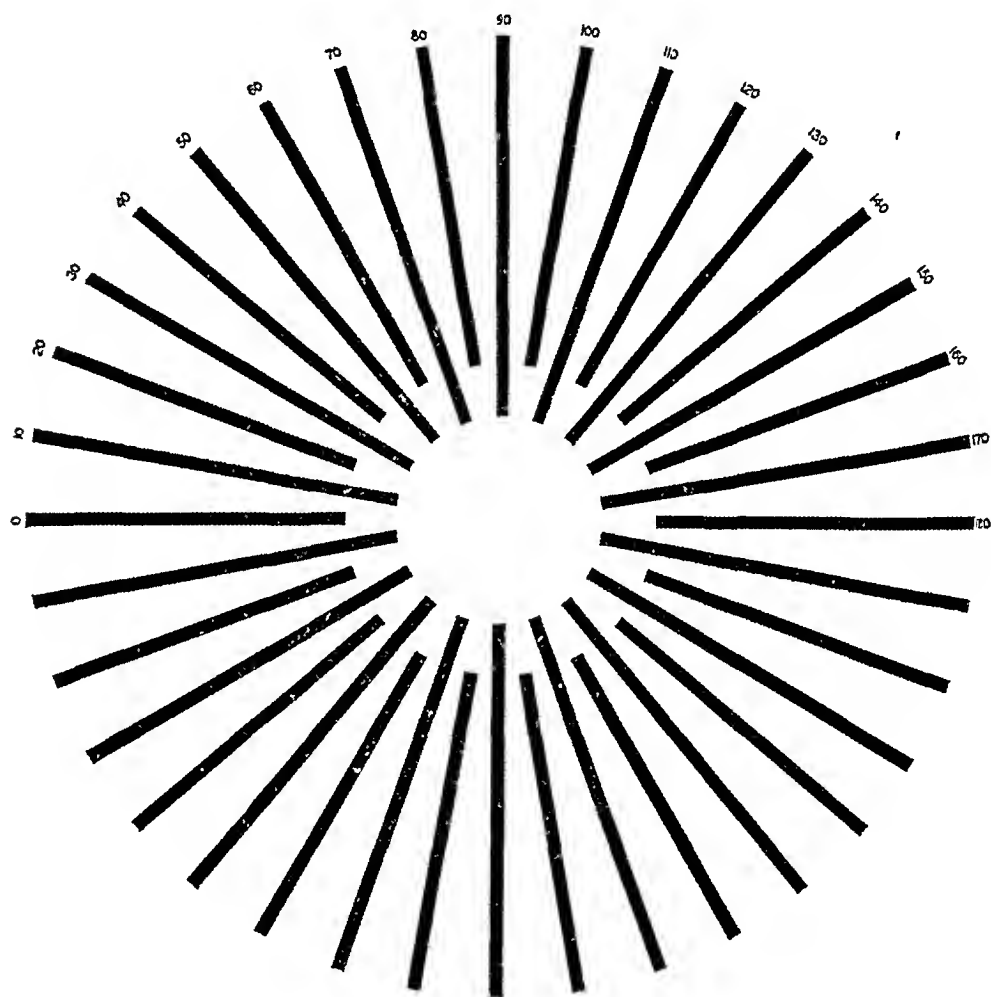


Fig. 1.—Astigmatic dial 1, with thirty-six radiating lines at intervals of 10 degrees.

being illuminated with flood-lights so that the space around the dials will be almost as bright as the dials themselves. A dark background should be avoided, for two reasons: First, it is desirable to have a volume of light entering the pupil such as will produce a size of pupil equal to that found under average working conditions—neither the large pupil produced by dim illumination nor the small pupil produced by overbright illumination is desirable. The larger pupil introduces into the problem the factor of refractive errors outside the visual zone of the

cornea. These are often substantial. The small pupil reduces the size of circles of diffusion in a way similar to a stenopaic disk, although, of course, to a less degree, thus obscuring the effects of astigmatism. Second, the retina responds more sensitively (as was shown by Cobb and Moss¹¹) when the surrounding field is such that there is not a marked contrast in brightness between the chart and the surrounding field.

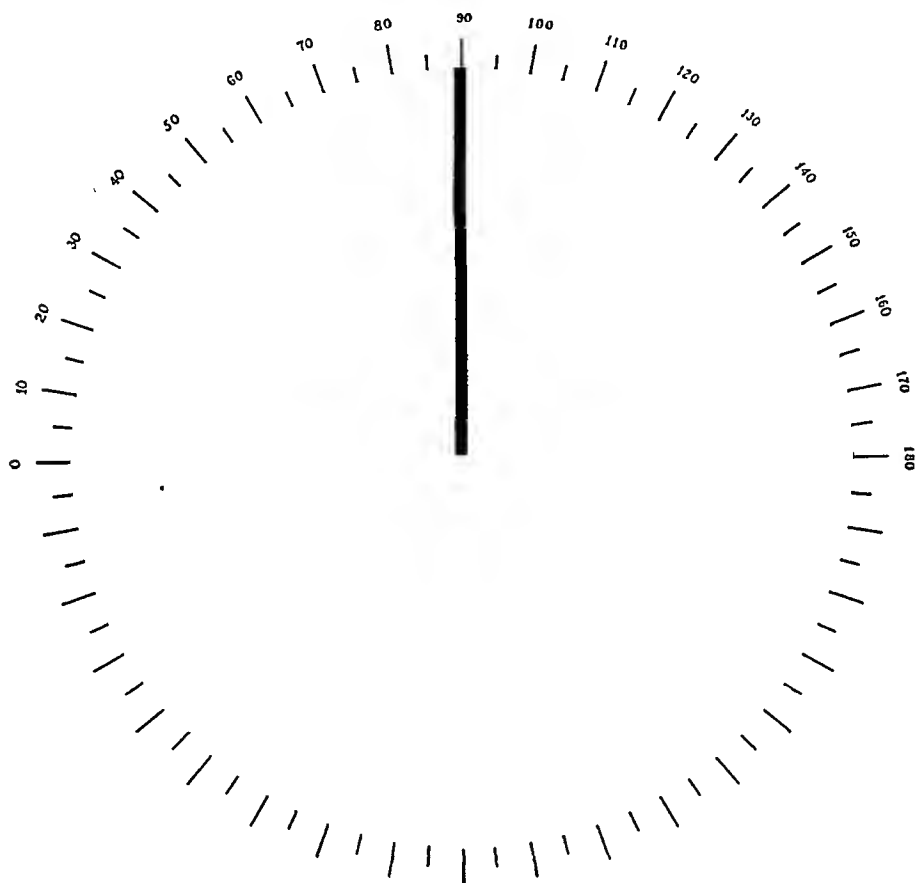


Fig. 2.—Astigmatic dial 2, with a cross adjustable at any angle.

The following experiments may be tried on one's own eyes if one has approximately normal vision—vision of 6/8 or better. If not, an intelligent patient may be used, preferably one with an accommodation of at least 4 D. and a pupil about 4 mm. in diameter.

One should start with the eye corrected to approximate emmetropia.

Experiments on Fogging.—A trial frame is placed comfortably over the glasses ordinarily worn. The eye not tested is occluded with

11. Cobb, P. W.: The Effect on Foveal Vision of Bright Surroundings, *J. Exper. Psychol.* **1**:540, 1916. Cobb, P. W., and Moss, F. K.: The Effect of Dark Surroundings on Vision, *J. Franklin Inst.* **206**:827, 1928.

an opaque disk. The smallest letters possible are read with the chart at 6 meters.

Experiment 1: A $+0.25$, a $+0.50$, a $+0.75$ and a $+1.00$ sphere are placed in the trial frame in succession; the smallest letters possible are read in each case, and a note is made of these. This will be referred to as "fogging."

This should show a definite reduction in vision for each addition of a plus sphere. If no definite reduction occurs with a $+0.50$ sphere, this shows that there is some uncorrected hypermetropia. An emmetropic eye with a $+0.50$ sphere reads about two lines less on a Green-Ewing chart.¹²

Experiment 2: The same procedure as that in experiment 1 is carried out, but a -0.25 , a -0.50 , a -0.75 and a -1.00 sphere are used.

This should show no definite reduction in vision if the subject has good active accommodation.

Experiment 3: Experiment 1 is repeated, a series of opaque disks with apertures of different sizes ranging from 5 to 2 mm. being used to reduce the size of the pupil. It should be found that the effect of fogging is markedly less with a small pupil.

Experiment 4: Experiment 1 is carried out on the other eye, and the findings are recorded. If the corrections worn by the eyes are well balanced, the reduction in vision will be about the same in each eye.

Experiment 5: Experiment 1 is carried out on both eyes simultaneously. In the average case the reduction in vision will be markedly less than when one eye is tested, showing that the subject relaxes his accommodation more when both eyes are tested together.

Experiment 6: Experiment 1 is repeated stronger lenses (up to $+4$ D.) being used, with the test object at 40 cm. (seen by the emmetropic eye with a $+2.50$ D. lens).

Experiment 2 is repeated, with the test object at the same distance (40 cm.), with stronger concave lenses (up to whatever power is needed to take up all the accommodation and produce some reduction in vision).

Experiments in Astigmatism.—Experiment 7: This experiment is like experiment 1, except that instead of observing the test letters the subject observes the astigmatic dial 1 (the dial with thirty-six radiating lines at intervals of 10 degrees).

If the subject sees all the lines alike—equally distinct or, when vision is fogged, equally blurred—either he has no marked regular

12. The great advantage of the Green-Ewing chart is that the test letters are graduated by geometric progression, making the ratio of difference in size the same with each successive line. For example, the letters on the 6/10 line are 25 per cent larger than the letters on the next line; these, in turn, are 25 per cent larger than the letters on the next line, and so on for the whole scale.

astigmatism which is uncorrected by his glasses or, if he has some uncorrected astigmatism, he is not a keen enough observer to detect it.

Experiment 8: (a) A plus sphere is placed in the trial frame over one eye (the other eye being occluded with a blank disk); a sphere which will fog the vision to 6/10 or 6/8 should be chosen.

(b) A -1.00 cylinder, axis 90 is added, and the appearance of the astigmatic dial is observed and recorded. The line at 90 degrees and one or possibly two lines on each side of it are much darker, more distinct and better focused than any of the others.

(c) The axis of the minus cylinder is changed from 90 degrees to various other degrees, and note is taken of how clearly lines in the chart correspond.

(d) Experiment 8 *b* and *c* is repeated, -0.75 , -0.50 , -0.25 and -0.12 cylinders being used.

Experiment 9: Experiment 8 is repeated with less fogging and without fogging.

Experiment 10: Experiment 8 is repeated, -0.25 , -0.50 and -0.75 spheres being used. It is noted that with the conditions as in experiment 8, i. e., with vision fogged, the chart shows the clear lines in the same axis as the minus cylinder. But under the conditions of experiment 10 there may be no marked difference in the radiating lines with a cylinder which is easily detected when the vision is fogged; then, as the minus sphere is increased, the time comes when the clearest lines are at right angles to the axis of the cylinder. With a little practice and by selecting the proper combinations of a plus sphere and a minus cylinder at different axes, the subject can make different axes come out clear at will; e. g., with a minus cylinder, axis 45 he can make the lines at 45 degrees clear, or by changing his accommodation he can bring out the lines at 135 degrees.

This shows how worthless the astigmatic charts are for finding the axis of astigmatism unless the accommodation is cared for by proper fogging.

It should be noted also that with a small amount of astigmatism no difference in the radiating lines is detected if the fogging is too great. The ideal fogging is produced by the sphere which fully corrects the hypermetropia plus another sphere which equals the amount of the astigmatism; e. g., if the true refraction is $+1.00$ sphere $\ominus +0.75$ cylinder, axis 80, the best fogging to show the astigmatism by the dials is obtained with a $+1.75$ sphere.

Hence, when the amount of astigmatism has been determined in a given case, it is often wise to corroborate the axis by placing in the trial frame the sphere which equals the stronger axis in hypermetropia,

and that which equals the weaker axis in myopia, and determine the axis of astigmatism again on dial 1.

Experiment 11: In a series of cases of astigmatism, either natural or produced by suitable spherocylindric lenses, the axis is first determined by the method just described and then the amount is measured by use of dial 2, the dial with the cross adjustable at any angle.

The cross is set at the angle determined by the test with dial 1. One line will be clearer than the other. This is the axis of the plus cylinder required. The other line will be less clear. This is the axis for a minus cylinder to correct the astigmatism. Minus cylinders are tried at this axis until the one is found which makes the two lines alike (not the one which makes this line as distinct as possible, for this would over-correct the astigmatism). When the correct power is found the two lines of the cross will be equally distinct. (If a strong minus cylinder is required, the plus sphere must be made stronger to maintain the fogging.)

The first estimate of the amount of astigmatism, or, in other words, of the strength of the cylinder required to correct it and make both meridians equal, has been made. One is now ready to measure the spherical error more accurately, after which the amount of astigmatism will be verified and a still more accurate measurement made.

The fogging is reduced until the maximum vision is found. A power such that the addition of a $+0.50$ sphere reduces the vision from $6/5$ to $6/8$ is selected. The effect of a $+0.25$ cylinder and then that of a $+0.12$ cylinder is tried, with the axis first in line with the correcting cylinder and then at right angles to it. Unless the hypermetropia of the subject is not fully corrected, he should promptly and consistently tell which way the cylinder is held. The cylinder held at either axis will blur the axis on the chart, thus making the opposite axis the more distinct one.

SOME DIFFICULTIES ENCOUNTERED

When vision is adequately fogged, a subject, after viewing dial 1 for several minutes, often insists that all thirty-six lines look alike. If a -0.50 cylinder, axis 90 is held before the eye, he may respond that the lines are still alike. If the cylinder axis 180 is held before the eye, the subject may respond that the horizontal lines are blackest. His attention should be directed to dial 2, with the lines vertical and horizontal, and he can easily see that the horizontal line is blacker. Minus cylinders, axis 90 are inserted until both lines appear equally black; one should not be surprised to find that frequently astigmatism of 0.50 D. or more exists. Of course, when the amount of astigmatism is determined in this manner great care must be exercised in determining the axis, and in such cases I suggest testing the patient again with dial 1.

Rarely, the subject will see no difference in the lines with a -0.50 cylinder in any position, and the response is that all the lines on dial 1 look double or shadowed. If so, his vision may be overfogged—an error which may occur when the astigmatism is slight. Some persons will select lines in two directions—not at right angles. In these cases the astigmatism is slight and somewhat irregular. The subject is using a special part of the focal pencil which nowhere, in his case, brings the light to a perfect point focus. He chooses the place which gives him the best results in seeing.

ANOTHER DIFFICULTY

The subject selects the blackest lines on dial 1 and after the cross-dial (dial 2) is set, minus cylinders up to, say, -0.75 are inserted. At this point the response may be: "Both lines appear double, but equally black." The most frequent cause of this is fogging. One must make sure of this by holding a minus sphere before the eye. This usually removes this doubling. If not, the axis is tried again with dial 1. There are two ways of selecting the axis at this point: The sphere is reduced sufficiently to give vision of $6/6$; then the minus cylinder is removed from the frame, and the subject's attention is directed to dial 1. The center of the group of black lines may now appear to be 5° or 10° degrees on either side of that originally selected by the subject. The cross-dial is set at the new axis. The spherical power is increased to refog vision; the -0.75 cylinder is inserted, and one proceeds toward accurate measurement of the amount of astigmatism. Another method is to reduce the sphere until the patient has vision of $6/5$ and rotate the cylinder while the patient views letters on the $6/8$ or $6/6$ line. The axis to choose is the one which gives the best focus of these letters.

The astigmatic dials mentioned in this article are manufactured by the E. B. Meyrowitz Surgical Instruments Co., Inc., 520 Fifth Avenue, New York.

p_H AND BUFFERS IN RELATION TO OPHTHALMOLOGY

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PHILADELPHIA

The determination of p_H ¹ has proved of great value in the perfection of many processes in various important industries. The usefulness of this test in the canning of fruits and vegetables has paralleled its application in the baking, tanning and sugar industries. Manufacturers of pharmaceutical products, those engaged in the dairy industry and many others employ p_H for one purpose or another in the betterment of their products.

In the basic sciences, such as bacteriology, biology and pathology, as well as in dentistry the use of p_H is well established. Thus it has been noted that there is a definite relationship in cow's milk between the p_H and the content per cubic centimeter of leukocytes and streptococci.² The importance of knowing the p_H of culture mediums and the rôle of p_H in bacterial growth is well known. Studies on the p_H of saliva in relation to the decay of teeth as well as in a number of physiologic and pathologic conditions have contributed much to the knowledge of physiology, bacteriology, immunology, pathology and other branches of medicine.

In this article, I have attempted to study p_H further as it relates to the problems and concerns of ophthalmology. Before results of these studies with p_H and also with buffers are stated, some consideration should be given to the method of approach and to the underlying principles behind the applications of p_H and buffers in ophthalmologic work.

The control of the p_H of a solution or ointment is of value, since it gives an accurate quantitative reading of the acid, alkaline or neutral reaction. The p_H bears no relation whatever to the osmotic pressure. Its determination is evolved around the theory of Arrhenius, wherein it is assumed that when an acid or an alkali is dissolved in water it breaks into ions (atoms) or groups of the same, which contain positive or negative charges of electricity. For example, water (H_2O) will be

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1. The p_H is the logarithm of the reciprocal of the hydrogen ion concentration. It may be defined as the power of 10 which indicates the fraction of a gram of hydrogen ions per liter of solution.

2. Baker, J. C., and Van Slyke, L. L.: A Method for the Preliminary Detection of Abnormal Milks, Bulletin 71, New York Agricultural Experiment Station, June 1919.

electrolyzed into H (+ ions) and OH (— ions). Determination of p_H is considered a more refined and delicate test than is a test with litmus.

A solution definitely shown to be acid or alkaline by determination of the p_H may sometimes give a neutral reaction to litmus. Titration gives the amount of acid or alkali in a solution. It does not indicate the intensity or the degree of the acid or alkaline content of the solution. A striking example of the information to be obtained from titration and p_H was given by LaMotte.³ Three acids—hydrochloric (corrosive), acetic (used in vinegar), and boric (used in eye washes)—when taken in equally concentrated solutions in a tenth-normal titration bear a relation of acid intensity in the order of 15,000:200:1. These three acids, respectively, with the same value of tenth-normal titration show, however, different values for p_H —1, 2.9 and 5.2, respectively.

By using determinations of p_H Nakashima⁴ found the hydrogen ion concentration of the dark-adapted eye (retina) to be p_H 7.3, while in the illuminated eye it is p_H 7 or less.

The p_H scale as devised by Sorenson is similar to that of the metric system, wherein the division of p_H measurements is in multiples of 10. It is illustrated by figure 1, in which the apex of the two sides of a triangle at 7 represents the neutral point, or p_H 7. From this point, on one side of the formed angle centimeter divisions represent successive alkaline increase from p_H 7 to 14, while on the other side divisions from p_H 7 to 1 indicate definitely increasing acidity.

Each millimeter measurement on the scale in the illustration represents the fraction of p_H . Thus, p_H 4.5 is represented at 4.5 cm. on the scale; p_H 6 is 10 times as acid in reaction as p_H 7, p_H 5 is 100 times as acid as p_H 7, and so on, in multiples of 10 for the acid or alkaline side of the scale. The value p_H 7, or neutrality, is supposed to be represented by that of pure water.

METHODS OF OBTAINING MEASUREMENTS OF p_H

There are two methods of checking p_H , viz.:

1. The colorimetric test, which is a comparative color test. In this test indicators which are very sensitive to small ranges of p_H are used. A set of about a dozen would be sufficient for work in ophthalmology. Clark,⁵ Lubs and others have prepared sets of these dyes which are commonly used. In this test the unknown solution, with the indicator

3. LaMotte, F. L.: *The A B C of p_H (Hydrogen Ion) Control*, ed. 10, Baltimore, Waverly Press, Inc., 1934, p. 21.

4. Nakashima, M., in Krause, A. C.: *The Biochemistry of the Eye*, Baltimore, Johns Hopkins Press, 1934, p. 85.

5. Clark, W. M.: *The Determination of Hydrogen Ions*, ed. 3, Baltimore, Williams & Wilkins Company, 1928, p. 411.

added, is compared with a series of stable buffered solutions of known p_H . When the color of the unknown and that of the known buffered indicator are exactly matched, the p_H of the unknown solution will be identical with that of the known control solution.

A modification of this colorimetric test is similar to the technic just described, except that papers saturated with indicators are used instead of tubes containing the buffer solutions used as indicators. A drop of the unknown solution is placed on the bibulous indicator paper, and this is compared with a color chart until the proper matching of color is noted. The p_H is then recorded. Another variation of this test is the

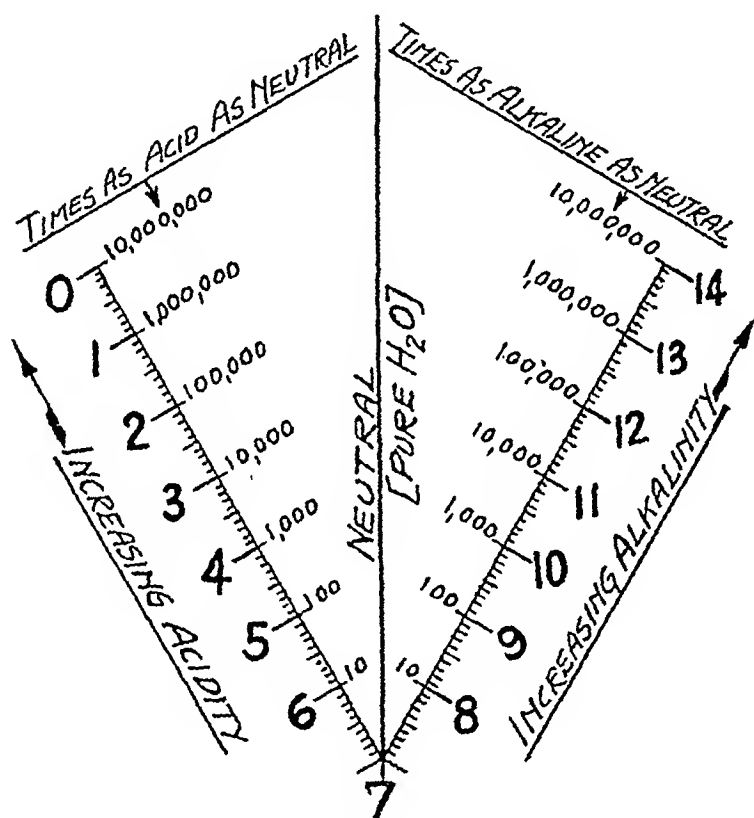


Fig. 1.—Diagram showing increase of acidity and alkalinity by logarithmic progression.

use of an indicator in the unknown solution. The resultant color is compared with the master color chart as already noted. The modifications just given are of value for the rough determination of p_H . The readings are obtained quickly, but small variations in p_H cannot be ascertained. Indicators and standard color charts are now available through a pharmaceutical concern.

2. The method I used for about a year is the electrometric or potentiometric test. Several concerns make the necessary apparatus. This method is accurate, and the technic is simple. It enables one to ascertain fractional differences in p_H .

The instrument I use contains a double stand (*A*) with an upper shelf holding three tubes for testing the p_H of alkaline solutions and a lower shelf holding a like number of tubes for the examination of the p_H of acid solutions. The middle tube of both sets contains a super-saturated solution of potassium chloride in distilled water. A buffer known to have a p_H of 7 is placed to the right of the solution of potassium chloride for examination of the alkaline solution to be tested. For testing an acid solution I used a buffer known to have a p_H of 5. The empty bottle to the left of the solution of potassium chloride on each shelf is used for the unknown solution. The buffer known to have a p_H of 7 was devised by Sorenson; the solution with a p_H of 5 was suggested by McIlvaine. When an acid solution with a p_H up to 7 is to be tested,

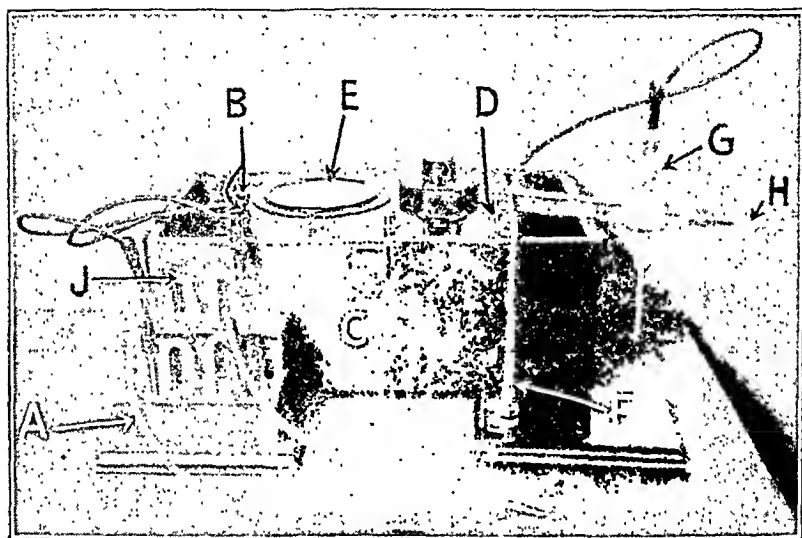


Fig. 2.—Potentiometer for the electrometric determination of p_H .

a pinch of quinhydrone powder is stirred into both the solution of unknown p_H and the buffer solution known to have a p_H of 5. Platinum terminals placed in both the solution of known p_H and the solution of unknown p_H are connected by leads to their respective posts (*B*) in the potentiometer (*C*).

U tubes (*J*) containing agar saturated with potassium chloride serve as conductors between the tube containing the solution of known p_H , the middle tube containing saturated solution of potassium chloride and the tube containing the solution the p_H of which is to be tested. In the testing of alkaline solutions no quinhydrone is used, and the terminals are antimony electrodes. The U tubes are used in the examination of alkaline solutions, as in the aforementioned procedure for the testing of acid solutions. Only one solution of unknown p_H , either acid or alkaline, can be tested at one time. The switch *D* opens the circuit, and

the p_H is quickly read on the dial (E). The aforementioned tubes are suitable when one has a fairly large amount of solution to work with—at least a few cubic centimeters of the unknown solution.

When only a small quantity of the solution of unknown p_H is available, as in the examination of tears, a Cullen tube (G) must be used. The Cullen tube⁵ is held upright by the stand F . In the center of this tube is a removable platinum wire, to which, when it is moistened with distilled water and immersed in quinhydrone powder, the powder adheres. The Cullen tube is now ready for use in making a determination of p_H . The platinum wire with the holder is inserted into the pipet portion of the Cullen tube and the tears of unknown p_H can be sucked in by the slow withdrawal of the arm (H). There is a minimum of exposure of the tears to the air. Care must be taken that no quinhydrone drops from the platinum wire into the eye, for if this occurs the eye will for a short time become red and irritable. The test for p_H with the Cullen tube is simple and finds its greatest usefulness in cases in which only very small quantities of solution (1 or 2 drops) can be obtained. The Cullen tube with the solution of unknown p_H is then inserted into the supersaturated solution of potassium chloride. A U tube containing agar saturated with potassium chloride connects the solution of unknown p_H in the Cullen tube in the saturated potassium chloride solution with the buffer solution of known p_H 5, to which a pinch of quinhydrone is added. The leads of the Cullen tube and platinum terminal in the buffer solution of known p_H 5 are connected with the potentiometer. The current is switched on, and the p_H of the unknown drop or two is quickly indicated on the dial of the potentiometer, as in the examination of large quantities of unknown solution. Protein and salt "errors" as well as dichromatism, which commonly occur in the colorimetric test, are not a factor in potentiometric determination. Turbid mixtures and ointments can be easily examined by the electrometric method. In both the colorimetric and the potentiometric examination care should be taken that the temperature of the solution of unknown p_H is about room temperature. This is necessary because p_H is the amount of ionization of a mixture, and this ionization is increased with heat. The buffer with a p_H of 5 may be used for testing solutions the p_H of which ranges through the values for acid and up to values for alkali of about p_H 8. The buffer standard solution of p_H 7 is best used for alkaline solutions with a p_H of from 7 to 14.

BUFFER SOLUTIONS

A buffer solution is one the known p_H of which is not materially affected by a reasonable addition of weak acid or alkali, i. e., a solution which is resistant to alterations in its p_H . Any solution may be strongly

or weakly buffered. Buffer solutions are sometimes made by including an acid with the salt of that particular acid, such as boric acid and the borates, or citric acid and the citrates. LaMotte, Kenny and Reed,⁶ as well as other authors of textbooks on p_H , have given a list of buffers of this type and others. A well known human buffer solution is blood. Human blood is buffered⁷ in part by virtue of the presence within it of phosphates, carbonates and bicarbonates. MacLeod⁸ quoted Van Slyke as stating that the normal range of the p_H of the blood is between 7.3 and 7.5. At p_H 7, coma occurs; at p_H 7.8, tetany sets in.

As has been mentioned previously, all the indicator solutions used in the colorimetric test are made with buffer solutions, and the solutions with p_H 5 and those with p_H 7 used in the potentiometric test here described are also buffered solutions. These solutions are reasonably stable and will retain their p_H for some time. A striking example of this stability is shown by a patient whom I treated. She used half of a 2 ounce (59 cm.) bottle of buffered eye wash the p_H of which was 7.6. She felt relieved and then put the medicine aside. When she returned after eleven months for refraction she asked whether the same medicine could be used again. I rechecked the p_H of the solution. It was 7.58.

Regardless of what ingredients are employed in making the buffer solution, great care must be used. The drugs should be weighed accurately, and the solution should be dated. The work should be entrusted to a careful technician or pharmacist. It is advisable, in making medicines for the eye, that the p_H of the buffer be clearly stated on the bottle in much the same way as is done by manufacturers selling nutrient mediums to bacteriologists. Only in this way can one check the p_H stability of preparations whenever desired.

TEARS

Hosford and Hicks⁹ quoted Brown as showing tears to be sufficiently buffered to permit a dilution of from 1 to 15 with neutral distilled water without altering its p_H . I have been fortunate in being able to verify the findings of Brown. A patient with glaucoma whose tears had a p_H of 7.15 was the subject. Distilled water of p_H 6.8 was instilled drop by drop into the eye. After the instillation of each 2 drops of

6. LaMotte, F. L.; Kenny, W. R., and Reed, A. B.: p_H and Its Practical Application, Baltimore, Williams & Wilkins Company, 1932, p. 20.

7. Remington, J.: Practice of Pharmacy, Philadelphia, J. B. Lippincott Company, 1936, p. 544.

8. MacLeod, J. J. R.: Physiology and Biochemistry in Modern Medicine, St. Louis, C. V. Mosby Company, 1930, p. 625.

9. Hosford, G. N., and Hicks, A. M.: Hydrogen Ion Concentration of Tears, Arch. Ophth. 13:18 (Jan.) 1935.

water the p_H was determined. Not until the addition of the eighteenth drop of distilled water did the p_H fall to 7.13.

About fifty eyes were examined for the express purpose of finding the p_H of tears. The subjects were patients who suffered no particular ocular discomfort but who came on account of a refractive error. The p_H ranged from 5.2 to 8.35. The greatest number had a p_H between 7 and 7.4. Tears of an equal number of eyes of patients suffering from extra-ocular diseases such as acute and chronic catarrh, dacryocystitis, foreign body, trachoma and gonorrhea showed almost the same range of p_H , the value being in the neighborhood of 7 in the majority of cases. The hydrogen ion concentration was not typical for any disease examined. The tears of both eyes in any one patient in health or disease were not always of the same p_H , nor was the p_H the same for the same eye when the p_H of the tears was rechecked at a later date. Tears were examined at different times of the day.

I was again able to verify the findings of Hosford and Hicks⁹ as to the wide range of the p_H of the tears of persons with gonorrhea. Even in cases of vaginal gonorrhea¹⁰ the p_H varied between 4.4 and 7. This wide divergence in p_H might be, as was pointed out, the cause for the great resistance to cure which patients with the disease show, since the gonococcus can grow in mediums the p_H of which is not necessarily fixed within narrow limits. I was able to observe, in the study of the p_H of tears, only one case of gonorrheal ophthalmia at the very onset of the disease. The tears showed a p_H of 7.1 for the diseased eye and a p_H of 7 for the normal eye.

It is important to remember in examining the p_H of tears that an accurate test is obtained only when there is a natural flow, i. e., when the lacrimal apparatus is not artificially stimulated. The method which I have utilized when tears were not easily obtained consisted in instilling 1 or sometimes 2 drops of distilled water of known p_H into the lower cul-de-sac. A well is created by the patient gently pulling forward the lower lid. The patient is told to rotate the eye in all meridians. The tears are sucked into the pipet of the Cullen tube and are examined with the potentiometer. Whenever distilled water is used, the p_H of the water must be known to make sure that the tears and not the water is being examined. When in doubt a reexamination should at once be made with another drop or two of water to see whether the p_H varies. It is important to know the p_H of the distilled water used.

The use of lens paper to absorb tears and then examination by the colorimetric test is not as accurate a method. Indeed, I have immersed lens paper in distilled water and have found an increase in alkalinity

10. Nine patients from the clinic for patients with genito-urinary diseases of St. Christopher's Hospital for Children were examined.

of the water from p_H 6.5 to 7. It is important to bear in mind when examining tears for p_H that the patient has not been using medicaments in the eyes for some time, since prolonged use of ophthalmic medicines may alter the p_H of the tears.

DISTILLED WATER

In figure 1, p_H 7 was supposed to represent the p_H of neutral distilled water. However, such an assumption is not absolutely correct in all cases, since the p_H of distilled waters may differ. Gifford ^{11a} quoted Truog as giving 5.8 as the p_H of distilled water. I examined fifteen samples of distilled water obtained from an equal number of drug stores, and obtained the following readings for the p_H of the specimens by potentiometric tests, in the order obtained: 5.18, 4.88, 5.78, 5.9, 6.18, 6.2, 7.1, 7, 7.48, 6.1, 6.6, 5.9, 6.5 and 5.65. The fifteenth specimen was triple-distilled water in a sealed ampule. It had a p_H of 7.1 and when left exposed for ten minutes showed a p_H of 6.8. One reason for the variation in p_H of the distilled waters examined may have been that the makers of the water exposed it to the air in order that the beverage might be more palatable. Water absorbs 0.03 per cent of carbon dioxide from the air; this increases its acidity and reduces its p_H beyond the neutral range of p_H 7. It is possible that the difference in p_H of the various distilled waters may account for the wide variation between the values for hydrogen ion concentration of Gifford and Smith ^{11b} and those for the hydrogen ion concentration of a set of the same ophthalmic medicaments (table 1, columns 1 and 2) compounded at two reliable drug stores. Column 1 of table 1 gives the ophthalmic medicines compounded by druggist A and dispensed in cork-stoppered bottles. Druggist B, using the drugs from the same manufacturer, dispensed the mixtures in bottles with screw caps (column 2). The third column gives the p_H of the same medicaments that were prepared in buffer solutions and placed in cork-stoppered bottles. This was done to show the relative value of the two methods of dispensing ocular medications (in bottles with cork stoppers and in bottles with screw caps).

The composition of the average medicine bottle, I have learned, contains too little alkali to be of serious consequence in affecting the stability of the p_H of the medicament. All the bottles in each group were opened daily for ten minutes and then closed until the following day. The purpose of this was to simulate the amount of exposure when the contents were used during a two month period. This method is not above criticism, since it exaggerates the use to which the contents of the bottles are put. It may be argued that a 1 ounce bottle opened so much every

11. (a) Gifford, S. R.: Reaction of Buffer Solutions and of Ophthalmic Drugs, *Arch. Ophth.* **13**:81 (Jan.) 1935. (b) Gifford, S. R., and Smith, R. D.: Effect of Reaction on Ophthalmic Solutions, *ibid.* **9**:227 (Feb.) 1933.

day for use would not last two months. I have purposely made the second reading of the p_H after subjecting the medicaments to more severe use than would ordinarily be expected of them. The fact was, however, that all the bottles were subjected to the same treatment whether they held buffered or unbuffered medicines. This method served only as a comparative means of testing the stability.

In table 1, the p_H in ordinary type is the hydrogen ion content just after the medicine was made; the p_H in boldface type is the value at the end of two months, after the bottle was opened daily for ten minutes. It will be noted that the p_H of the same preparation differs

TABLE 1.— p_H of Preparations Made with Distilled Water, Dispensed in Bottles with Cork Stoppers and in Bottles with Screw Caps, Compared with the p_H When a Buffer Solution and a Cork Stopper Are Used

Drug Used, 1 Oz. Mixtures	p_H of Preparations Made with Distilled H ₂ O by Druggist A, in Bottles with a Cork Stopper		p_H of Preparations Made with Distilled H ₂ O by Druggist B, in Bottles with a Screw Cap		p_H of Preparations Made with Buffer Solution in Bottles with a Cork Stopper	
	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.	First Reading	Reading After 2 Mo.
Atropine sulfate, 4 grains.....	5.75	5.40 M	6.20	5.10	7.55	7.12
Butyn, 2% solution.....	6.20	5.25 P	6.30	5.75	Not made	
Boric acid, saturated solution...	4.80	4.90 M	5.00	5.18	6.30	6.30 M
Boric acid-sodium borate solution	9.10	9.00 M	9.10	9.08	8.50	8.45 P
Cocaine, 2% solution.....	6.10	4.15 M	5.80	5.60 M	5.94	4.08
Physostigmine sulfate, 2 grains	6.15	6.50 M	6.90	5.70	7.50	7.30 P
Physostigmine sulfate, 1 grain..	6.10	6.20 M	6.20	5.56 M	7.50	7.63 P
Phenacaine hydrochloride, 1 grain	6.48	5.08 M	6.50	5.40	5.50	5.90
Homatropine hydrobromide, 1 grain	7.70	5.73 M	6.95	8.19	7.30	7.70 P
Metaphen, 1:10,000 dilution.....	8.95	7.88 M	8.87	7.81	7.58	7.20
Procaïne hydrochloride, 4% solution	5.70	6.50 M	5.80	6.12	7.16	6.18 P
Ethylhydrocupreine hydrochloride	6.20	5.95 P	Not obtained		Not made	
Pilocarpine, 1% solution.....	5.20	5.20 P	5.94	6.05	7.28	7.42 P
Silver nitrate, 4 grains.....	4.08	3.61 P	0.70	0.90	Not made	
Sodium chloride, 0.9% solution...	6.42	6.75 M	6.50	6.88 P	7.32	7.45 M
Zinc chloride, 1 grain.....	5.70	5.15 M	6.50	4.80	6.08	6.60 P

* In this table and table 2 M indicates mold and P, precipitate.

in all cases except one, that of the sodium baborate eye wash (made of sodium borate, camphor and water). It is interesting to note that in the case of each of the cork-stoppered bottles either mold or a precipitate developed in the medicine, as against only three bottles with screw caps. This formation of precipitates and mold was also noted to a great extent in the preparations made with the buffer solution. It was because of this undesirable sediment that I soon made the change to bottles with droppers, for dispensing ophthalmic medicaments. The change in p_H after two months of opening the bottles, as shown in table 1, was least in preparations made with the buffer solution, even though cork stoppers were used. It would seem, therefore, that the best way to dispense the medicaments is in buffer solutions and in bottles with screw caps or droppers.

BUFFER SOLUTIONS IN OPHTHALMOLOGIC PRACTICE

Buffer solutions are useful in ophthalmologic practice because they insure uniformity of product and are quite stable. They are, in the great majority of cases, more easily tolerated by the eye than are nonbuffered solutions. The buffered solution is a vehicle the p_H of which can be altered to suit the needs of the patient, the active drug contained always remaining the same. For example, when bacteria can thrive only in an acid medium, the buffer is made alkaline, or the reverse.

Since the publication on buffers by Gifford and Smith,¹¹ I have used the buffer solutions which they recommended, with satisfactory results. Their combination buffer solution is a modification of that suggested by Atkins and Pantin¹² in 1926, being a buffer solution used for the testing of the p_H of sea water. For about a year, however, I have used exclusively a buffer solution recommended by Palitzsch.¹³ It is simply made and is well tolerated by the eye. Its virtue lies in the fact that it contains an acid and the particular salt of that acid. It contains three well known bland and familiar medicaments, each of which has been used singly or in combination in ophthalmology, viz., boric acid, sodium borate and sodium chloride. The boric acid acts at the same time as a preservative and a mild antiseptic. Two parent solutions are made. One is a twentieth-molar solution of sodium borate, containing 19.108 Gm. of sodium borate to the liter. The other is a fifth-molar solution of boric acid, containing 12.404 Gm. of boric acid with 2.925 Gm. of sodium chloride. By the admixture of various proportions of the two parent solutions a number of different buffer solutions suitable for vehicles are obtained. By adjusting different proportions of the two parent solutions Palitzsch made seventeen different vehicles with a p_H ranging from 6.77 to 8.69. Each vehicle has a therapeutic value because of the ingredients (boric acid, sodium borate and sodium chloride). The aforementioned combinations of buffer solutions can be used in dispensing all preparations except in the cases of butyn, zinc and cocaine or other substances which require definite acid vehicles. In prescribing these the mixture of a fifth-molar solution of boric acid with sodium chloride can be employed.

The most common buffer vehicles which I have made and used are as follows:

A buffer solution of p_H 6.77, made by combining 0.3 cc. of a solution of sodium borate with 9.7 cc. of a solution of boric acid and sodium chloride.

12. Atkins, W. R. G., and Pantin, C. F. A.: A Buffer Mixture for the Alkaline Range of Hydrogen Ion Concentration, *Biochem. J.* **20**:102, 1926.

13. Palitzsch, S.: Manuel pratique de l'analyse de l'eau de mer, *Bull. Inst. océanog.*, Monaco, no. 409, April 1922.

A buffer solution of p_H 7.2, made by combining 1 cc. of a solution of sodium borate with 8.5 cc. of a solution of boric acid and sodium chloride.

A buffer solution of p_H 8.69, made by combining 6.0 cc. of a solution of sodium borate with 4 cc. of a solution of boric acid and sodium chloride.

A 3 per cent solution of glycerin, as recommended by Smith,¹⁴ the boric acid-sodium borate buffer of p_H 7.2 being used.

I have taken the last-named four vehicles and have added to them different medicinal agents. In each case only one ophthalmic drug was mixed with the vehicle of known p_H . After the addition of the drug

TABLE 2.— p_H of Solution After Drugs Were Added to the Buffer Vehicles

Drug Used, 1 Oz. Mixtures	Boric Acid-Sodium Borate Buffer, p_H 6.77		Boric Acid-Sodium Borate Buffer, p_H 7.2		Boric Acid-Sodium Borate Buffer, p_H 8.69		3 per Cent Glycerin in Boric Acid-Sodium Borate Buffer, p_H 7.2; Final p_H , 7.00	
	Read- ing After 2 Mo.		Read- ing After 2 Mo.		Read- ing After 2 Mo.		Read- ing After 2 Mo.	
	First Read- ing		First Read- ing		First Read- ing		First Read- ing	
Atropine sulfate, 4 grains.....	6.90	6.60	7.33	7.00	8.70	8.00	6.91	6.69 M
Phenacaine hydrochloride, 1% solution	6.50	6.50	Not made		8.62	7.95	Not made	
Homatropine hydrobromide, 1 grain	6.90	7.40	7.31	7.70	8.65	8.20	6.90	7.48 M
Pilocarpine, 1 grain.....	6.00	6.05	7.18	7.10	8.05	7.68	6.60	6.90 M
Physostigmine, 1 grain.....	6.80	6.70	7.30	7.20	8.65	8.42	6.83	6.80 M
Physostigmine, 2 grains.....	6.74	6.60	7.25	7.01	8.62	8.22	Not made	
Metaphen, 1:5,000 dilution.....	7.00	6.56	7.42	7.13 P	8.70	8.90 P	7.20*	
Metaphen, 1:10,000 dilution.....	7.00	6.23	7.30	6.80	8.71	8.53	7.05*	
Zinc sulfate, 1 grain.....	7.09	6.78 P	7.09	6.78 P	8.32	8.11 P	6.85*	

* Congealed at 40 F. (5 C.).

the p_H was again determined. The results are given in table 2. The bottles were opened for ten minutes daily for a period of two months, as in the tests the results of which are shown in table 1. After this period the p_H was again determined. It is noted in boldface type to the right of the original p_H . The bottles used in all these tests had droppers. It will be noted from table 2 that on the addition of a 3 per cent solution of glycerin to the boric acid-sodium borate vehicle the p_H dropped from 7.2 to 7. It is seen that in the great majority of cases the solution resulting from the addition of the drug to the buffer vehicle tended to show a slight rise in p_H . The opposite tendency is noted to a slight extent in the vehicle of p_H 8.69 and in the vehicle of p_H 7 with a 3 per cent buffer solution of glycerin.

14. Gifford, S. R.: A Handbook of Ocular Therapeutics, Philadelphia, Lea & Febiger, 1932, p. 55.

After two months of daily opening of the bottles for ten minutes at a time to simulate the amount of daily exposure to air that might occur in the ordinary use of a medicament, only metaphen in a 1:5,000 dilution at p_H 7.13 and 8.90 showed a precipitate. As zinc and phenacaine hydrochloride require an acid medium, precipitations with these were not unexpected. Molds were found in all the mixtures of glycerin. The mixture of metaphen and glycerin congealed when the temperature of the room dropped to 40 F. (5 C.). This occurred also in the case of the mixture of zinc and glycerin.

Ordinarily, eucatropine (a 5 per cent solution) is said to require an acid vehicle for making a pure solution. I have been able to use a boric acid-sodium borate buffer of p_H 7 with this drug and yet have complete solubility. No precipitate formed on standing.

Tice,¹⁵ in a review of an article on the preparation of eye drops by Buchi and Baeschlin, reported their successful use of boric acid-sodium borate buffer solutions and their methods of also utilizing the parent mixture of boric acid to include those drugs not soluble in alkaline or neutral solutions.

The buffers of a p_H ranging from 7 to 8 have proved effective for solutions which I used with contact lenses. The burning sensation experienced with contact lenses after wearing them for a short period when using physiologic solution of sodium chloride (p_H 6.4 to 6.5) or Ringer's solution (p_H 7.45) is not noted so soon when a buffer solution of p_H 7 to 8 is used. Ringer's solution gives a better result than does physiologic solution of sodium chloride, but is not as good as the buffer solution. This was verified in a number of instances. In one outstanding case a patient who could not retain a contact lens for more than one hour when physiologic solution of sodium chloride was used or for over two hours when Ringer's solution was used was able to use the contact lens comfortably for ten hours when a buffer solution was used. I believe this increased tolerance was made possible, first, by the medicaments comprising the buffer solution and, second, by the fact that this solution can be so altered as to make its p_H one which is pleasant to endure.

That the p_H of the fluid in a contact lens does not change materially has been borne out by the experiments carried out in the following cases.

CASE 1.—M. S. had a conical cornea in each eye. Tears were examined. Those of the right eye had a p_H of 7.25, and those of the left eye, a p_H of 8.05. A contact lens was inserted on each eye, a buffer solution of p_H 7.3 being used. The eyes became irritable after ten hours. When the contact lens was removed, the fluid in the contact lenses was examined. The solution in the lens for the right eye showed a p_H of 7.33, and that in the lens for the left eye showed a p_H of 7.03.

15. Tice, L. F.: The Preparation of Eye Drops, *Am. J. Pharm.* **108**:308, 1936.

CASE 2.—A. K. had high myopia. Tears could not easily be obtained. A contact lens was inserted in each eye, a buffer solution of p_H 7 being used. After two hours the eyes became irritable. The p_H of the buffer solution in each eye was 7.12. At another date a second test was carried out on the same patient. The p_H of the tears, which were then easily obtained, was 7.25 for the right eye and 7.05 for the left. A contact lens was placed on each eye, a buffer solution of p_H 7.53 being used. The lenses were worn about three hours and were removed on signs of oncoming irritation. The solution in the lens for the right eye showed a p_H of 7.52 and that in the lens for the left eye a p_H of 7.76.

The use of physiologic solution of sodium chloride with contact lenses in a few cases did not change the p_H more than 0.5. Of course, if the contact lens could have been worn longer, as is the case when a buffer solution is used, the p_H might have shown greater variations.

After the contact lens was worn for a while the eye became irritable in much the same manner as though there was a foreign body in the eye. Many patients, after wearing the contact lens for some time, in addition to irritation, noted a blurring in vision. With the thought that there might be a difference in the refractive index of the various solutions used, which, in addition, might cause ocular fatigue and irritation, Dr. Arthur Osol, of the Philadelphia College of Pharmacy and Science, assisted me in checking the refractive indexes of three solutions. The following almost identical results were obtained for all three mediums: The refractive index of the boric acid-sodium borate buffer of p_H 7.2 was 1.33416; that of the physiologic solution of sodium chloride of p_H 6.5 was 1.33405, and that of the triple-distilled water of p_H 7.1 was 1.33246.

I have used buffered solution of the boric acid-sodium borate type in a few hundred cases of extra-ocular disease. Only two of the patients reported that the results were not satisfactory. I believe that if these buffers were adjusted to the particular patient this shortcoming would be remedied. Ordinarily a p_H of from 7 to 7.3 was used. The startling improvements which I have obtained in acute and chronic external ocular infections have amply repaid me for the trouble and painstaking care required in the making of the buffer solutions.

SUMMARY AND CONCLUSIONS

The value of p_H in industry and science is briefly considered. A graphic description of calibrating p_H as devised by Sorenson is given.

The chief methods of determining p_H are described briefly, with particular reference to the electrometric or potentiometric test. The means of studying the p_H of small quantities of solution (e. g., tears) by the use of the Cullen tube is given.

It was shown that distilled waters vary in p_H . Therefore the same medicaments made with different distilled waters were found to vary.

The p_H of the tears of the same person in either health or disease may be different from time to time. Nor is the p_H of the tears of the two eyes always the same. The average range of the p_H of tears is between 7 and 7.5. Persons with extra-ocular diseases do not show typical changes in the p_H of the tears.

Buffer solutions are defined, and various examples of those which may be used in ophthalmology are given. The advantages of dispensing ophthalmologic prescriptions in bottles with a screw cap or dropper tip as against cork-stoppered bottles are shown.

Preparations made with a buffer solution as a vehicle are more stable and more soothing to the eye than those made with distilled water, owing to the medicinal properties of the ingredients of the buffer solution. The buffer solutions are mildly antiseptic and contain preservatives. The change in p_H of buffer solutions caused by the addition of various drugs used in diseases of the eye has been shown after compounding the preparation as well as after the simulated use of such preparations for some time.

The better results obtained by using buffer solutions instead of physiologic solution of sodium chloride or Ringer's solution for contact lenses is noted.

Buffer solutions can be used in counteracting extra-ocular infection by creating a reaction of the tears which is inhibitive to the growth of bacteria.

Miss Evelyn M. Carpenter and Service B of the ophthalmologic department of the Protesant Episcopal Hospital gave the financial aid necessary to make this study, and B. H. Hoffstein, formerly associated with the Philadelphia College of Pharmacy and Science, assisted in solving some of the pharmaceutical problems.

LESIONS OF THE FUNDUS IN POLYCYTHEMIA

REPORT OF CASES

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Polycythaemia vera is generally regarded as a clinical entity caused by primary hyperplasia of the erythroblastic elements of the bone marrow. Its origin is unknown. It is seen infrequently by the internist and even more rarely by the ophthalmologist, as vision is seldom disturbed. Examination of the fundus, however, is likely to reveal early characteristic lesions which are of value in the diagnosis. In view of these considerations I have decided to present this subject, together with a brief report of a few cases.

Although the term polycythemia signifies an increase in the cellular elements of the blood, it is now generally applied to an increase in the number of red blood cells or erythrocytes; therefore the disease is often called erythremia. Vaquez¹ in 1892 was the first to describe polycythemia, in an article entitled "Cyanosis with Polycythemia," while William Osler² in 1903 presented the well known and important paper entitled "Chronic Cyanosis with Polycythemia and Enlargement of Spleen." Hence the disease is also known as the Vaquez-Osler disease. The lesion of the fundus was called by Ascher fundus polycythemicus. As early as 1859 Hermann Knapp³ presented before the Naturhistorisch-medicinischer Verein of Heidelberg a report of a case entitled "A Case of Hyperemia of the Retina with Choked Disk." U. Kümme⁴ stated that he considered this case one of polycythemia. More recently articles on this condition have been published by Julius Ascher,⁵ Edward Jackson,⁶ Carl Behr,⁷ G. E. de Schweinitz and Alan Woods⁸ and others.

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5. Ascher, J.: *Klin. Monatsbl. f. Augenh.* **53**:388, 1914.
6. Jackson, E.: *Tr. Am. Ophth. Soc.* **11**:363, 1906.
7. Behr, C.: *Klin. Monatsbl. f. Augenh.* **49**:672, 1911.
8. de Schweinitz, G. E., and Woods, A.: *Tr. Am. Ophth. Soc.* **23**:90, 1925.

Recent studies in hematology with reference to polycythemia will probably change some of the earlier diagnoses with respect to cyanosis retinae, a term which, according to Posey,⁹ is applied solely in connection with heart disease. The case reported by Baquis¹⁰ and that of H. H. Tyson,¹¹ both of which were published in 1908, might now be considered cases of fundus polycythemicus occurring in conjunction with congenital heart disease with associated secondary or compensatory polycythemia rather than cases of cyanosis retinae. The changes in the blood in both cases were typical of polycythemia; that is, the red blood cell count was over 8,000,000 and the hemoglobin content was over 100 per cent in each case. Both patients suffered from congenital heart disease associated with splenic enlargement. Carl Behr⁷ regarded the condition in these cases as polycythemia. Cyanosis retinae is due to general cyanosis found in cases of polycythemia, heart disease and many other conditions; for this reason I believe that it should not be used solely in reference to heart disease.

There are two types of polycythemia. The primary form, or polycythemia vera, the cause of which is unknown, is characterized by an increase in the number of red cells, the hemoglobin content and the volume of the blood and is accompanied by headaches, malaise, neurologic symptoms, general cyanosis and enlargement of the spleen. It generally occurs between the ages of 35 and 65. The secondary or compensatory form is usually due to congenital heart disease, although other causes are emphysema, stenosis of the pulmonary artery, and dehydration of the tissues (as in cholera and dysentery). A temporary form is found in persons living in high altitudes. It may occur at any age. The general manifestations of the secondary form of polycythemia are similar to those observed in the primary type, except that in the secondary type the cyanosis is apt to be more marked; the tongue and lips are more cyanotic; the finger tips are more clubbed, and the spleen is not usually enlarged. Either type may be associated with mild or severe ocular manifestations. At times it is possible to distinguish ophthalmoscopically between the primary and the secondary type, as reported by Engelking.¹³

Attempts to differentiate the ocular symptoms in polycythemia from those in cyanosis retinae have been reported in the literature. General cyanosis is due to deficient oxidation of the blood. This manifestation may be either mild or severe and may be associated with ocular lesions. If it affects the retina it is called cyanosis retinae and is confined chiefly

9. Posey, W. C.: *Tr. Am. Ophth. Soc.* **10**:634, 1905.

10. Baquis, E.: *Arch. f. Ophth.* **68**:177, 1908.

11. Tyson, H. H.: *Arch. Ophth.* **37**:555, 1908.

12. Footnote deleted.

13. Engelking, E.: *Klin. Monatsbl. f. Augenh.* **64**:645, 1920.

to the blood vessels, the surrounding tissue often not showing changes typical of cyanosis, owing to the light reflex from the retinal pigment. This cyanosis is sometimes most evident in the physiologic cup of the disk. In albinos with polycythemia a cyanotic appearance of the fundus could probably be more easily recognized on account of the lack of normal pigment. For severe cases in which other parts of the eye are involved Baquis has used the term cyanosis oculi. In some cases of severe congenital heart disease there is probably a compensatory reaction due to some interference with oxygenation, which stimulates the bone marrow and causes increased cyanosis with secondary polycythemia. I suggest that cyanosis retinae or cyanosis oculi should be considered in conjunction with other diseases rather than as an entity.

Another type of polycythemia is known as polycythaemia hyper-tonica, or Gaisböck's disease. This type is associated with arterio-sclerosis, cardiac disease and renal disease and is accompanied by high blood pressure without splenomegaly.

The characteristic blood picture in polycythemia consists of an average high increase in the number of red blood cells from about 7,000,000 to 10,000,000, in conjunction with an increase in the hemoglobin content from about 110 to 126 per cent or over. The color index is low (less than 1), and the volume and viscosity of the blood are increased.

The fundus sometimes appears normal, while at other times lesions exist, which may be mild or severe, depending on the severity of the disease and whether or not the patient has responded to treatment. The existence of a high red cell count of the blood, with a corresponding increase in the hemoglobin content, frequently determines the degree to which the fundus is affected and also the extent of the vascular changes in the conjunctiva and iris.

The vascular lesion of the fundus is a part of the general vascular disturbance, which is not of an inflammatory nature, as proved by Carl Behr⁷ in a report of ocular microscopic examination. The characteristic finding in the fundus in polycythemia is marked distention and engorgement of the retinal veins, which appear purplish. This distention is due mainly to an increase in the blood volume and a thinness of the venous wall. The change in color is caused by excessive replacement of oxygen by carbon dioxide. The resultant venous stasis is the basic factor in the causation of the ocular lesion.

Of the seven cases of polycythemia reported in this paper, five are cases of the primary type, and two are cases of the secondary type. In the latter type there were no fundic complications; the retinal veins were distended, slightly tortuous and bluish; the retinal arteries were moderately dilated and were purplish. In one case the disk appeared reddish and the fundus had a grayish tinge; in the other there was a

bluish appearance of the physiologic cup and the veins, while the color of the fundus seemed normal. In both cases the ocular and tarsal conjunctivae were congested and their veins dilated. Slit lamp examination showed that in one case the iris was normal and that in the other there were dilated iritic blood vessels with chocolate-colored areas and dustlike pigment scattered over some of the vessels. Vision and ocular function were normal in both cases.

Of the cases of the primary type, in two there were lesions of the fundus, while in three the lesions were solely vascular, consisting of distention of the retinal veins, which were purplish and moderately tortuous. In these three cases the arteries appeared normal in caliber and color, and the disk was also normal. The fundic complications in the two cases of primary polycythemia were as follows: One patient presented venous engorgement in one eye, with edema of the disk and retinal hemorrhages; the other showed bilateral postneuritic atrophy of the optic nerve, with distended veins which were bordered by a distinct broad whitish band along the perivascular space, due to a transudation of plasma. The perivascular space of the retinal arteries was not involved.

At the meeting the Section of Ophthalmology of the New York Academy of Medicine in 1918 I¹⁴ presented a case of polycythemia complicated by unilateral thrombosis of the central retinal vein.

In the cases reported in this paper, vision was normal with each type of polycythemia, except in one case of the primary type. Visual disturbances were reported in 1919 by Friedenwald,¹⁵ who expressed the opinion that the deterioration of vision noted in those of his patients who were without lesions of the fundus was caused by retrobulbar neuritis and papillitis.

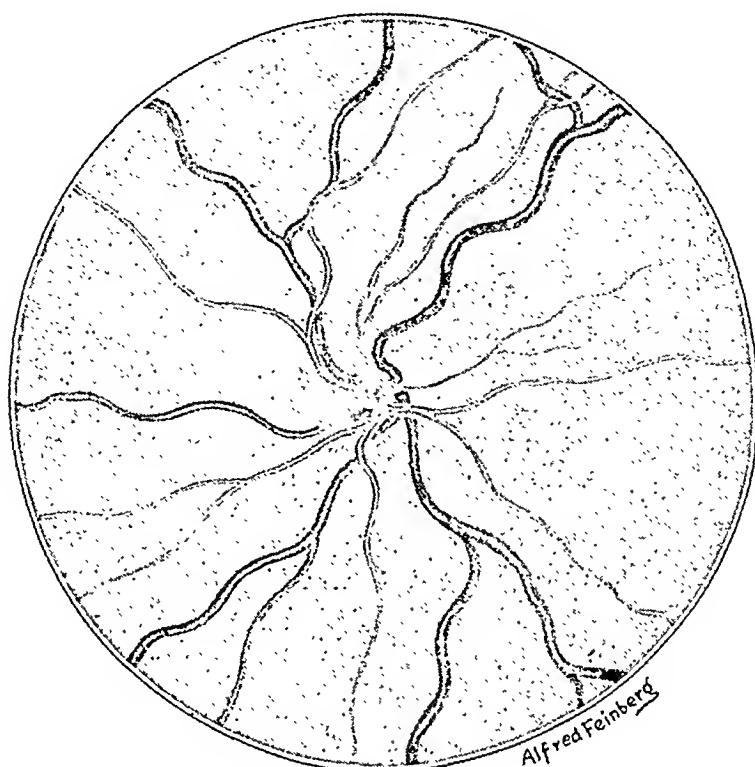
It should be remembered that variations and congenital anomalies, as well as local lesions, may be present in the fundus. The general symptoms of the disease will aid in the diagnosis of the condition of the fundus.

Carl Behr⁷ in 1911 published a report giving the pathologic findings in polycythemia, with illustrations of the distended and engorged retinal and uveal vessels filled with numerous red and a few white blood cells. He noted also edema of the disk but no inflammatory reaction.

The prognosis of polycythemia depends on whether one is dealing with the primary or the secondary type. In cases of the former type the prognosis depends on the severity of the disease, some patients living to an advanced age. Death is caused by complications such as

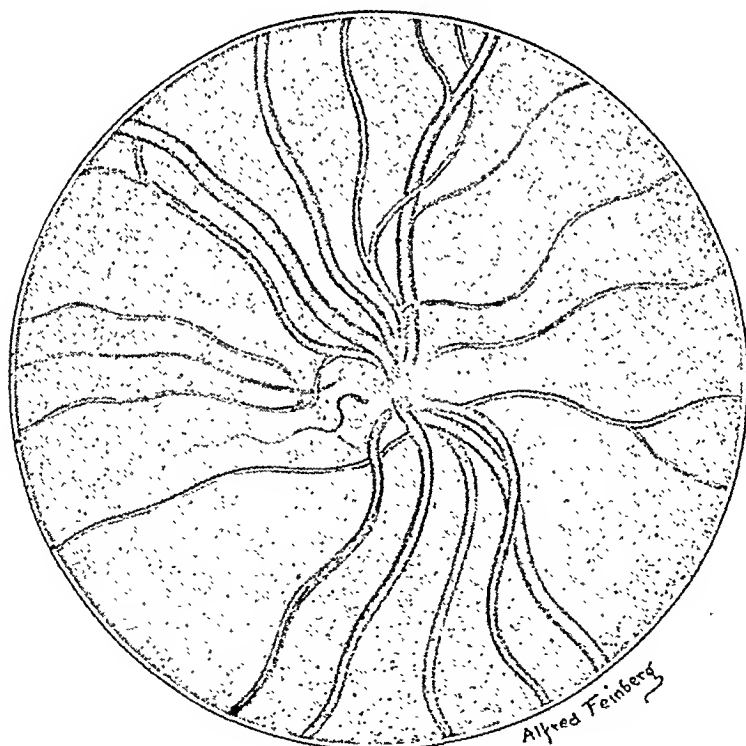
14. Cohen, M.: *Arch. Ophth.* **47:2**, 1918.

15. Friedenwald, H.: *Visual Disturbances in Polycythemia Vera*, in *Contributions to Medical and Biologic Research*, dedicated to William Osler, New York, Paul B. Hoeber, Inc., 1919, vol. 1, p. 495.



Polycythemia
(Primary)

Fig. 1.—Lesion of the fundus in primary polycythemia.



Polycythemia
(Secondary)

Fig. 2.—Lesion of the fundus in secondary polycythemia.

heart failure, hemiplegia, thrombosis and hemorrhages. In cases of the secondary type the prognosis depends on the severity of the cardiac lesion or other complicating disease.

Treatment differs for the two types. In cases of the primary type several methods of treatment have proved of definite value, including venesection, the use of phenylhydrazine and irradiation. In cases of the secondary type the causative disease is treated along the lines indicated by the condition.

Since few cases of polycythemia have been observed at the medical clinic and service of the New York Post-Graduate Medical School and Hospital, my associates and I were obliged to obtain additional patients for presentation. A brief report of these cases, including the average findings of the blood, follows.

REPORT OF CASES

CASE 1.—H. S., a man aged 64 years old, a factory worker, was admitted to the medical clinic of the New York Post-Graduate Medical School and Hospital on Feb. 27, 1932, complaining of vertigo. The history revealed no abnormality except neuralgic pains. The skin had been cyanotic for seven years. The patient seemed well nourished and did not appear to be acutely ill. The mucous membrane of the mouth and pharynx was bluish. The heart was normal in size; its sounds were of good quality, and there were no murmurs. The lungs were clear. The spleen and liver were moderately enlarged. The blood pressure was 126 systolic and 64 diastolic. The urine was normal. The red cell count of the blood was 9,040,000, with 130 per cent hemoglobin; the white cell count was 7,900. Roentgenograms of the long bones showed atrophic changes compatible with dyscrasia of the blood. Microscopic examination of the capillaries of the nail beds of the fingers showed them to be markedly dilated and packed with red cells, especially in the venous segment. The visual disturbances present were due to an incipient cataract in each eye. The findings for the fundus were: retinal venous distention with engorgement, the veins appearing purplish, and irregularity of the lumen of the superior temporal vein. The arteries and the disk were normal. Acetylphenylhydrazine was used in treatment, with remarkable results. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 2.—A. B., a married woman 65 years old, was first examined at the New York Post-Graduate Medical School and Hospital in 1929, at which time she complained of vertigo. The following were noted: cyanosis of the hands and lips, of twelve years' duration; clubbing of the fingers, and pain over the heart, which was enlarged (a secondary condition). The urine showed occasional red blood cells. The red cell count of the blood was 8,152,000 with 110 per cent hemoglobin. The blood pressure was 196 systolic and 112 diastolic. The spleen and liver were enlarged. Vision was normal in both eyes with correction, and the field of vision was normal. Examination of the fundus showed distended and purplish veins; the arteries and disk were normal. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 3.—O. C. M., a salesman 48 years old, was admitted to the New York Post-Graduate Medical School and Hospital on June 28, 1932, complaining of precordial pain (not radiating to the arms) and insomnia. The past history was negative. The face was deep red, and the mucous membranes had been purple for four years. The lungs were clear, and the heart was normal. The spleen was palpable. The blood pressure was 108 systolic and 82 diastolic. The red cell count of the blood was 8,000,000, with 154 per cent hemoglobin. The color index was 0.96. The oxygen capacity, oxyhemoglobin content, viscosity and blood volume were determined and considered as positive factors for the diagnosis of polycythemia. Vision in the right eye was 20/200 (the poor vision was due to congenital amblyopia); vision in the left eye was corrected to 20/20 with glasses. Examination of the fundus showed distended and purplish retinal veins, normal retinal arteries and a reddish disk. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 4.—F. M., a man 34 years old, was admitted to the Manhattan Eye, Ear and Throat Hospital (in the service of Dr. David Webster) in January 1934. There was a history of poor vision and cyanosis for three years. The chief complaints at the time of admission were poor vision, and varices in both legs. Physical examination showed marked cyanosis of the face. The liver and spleen enlarged. Study of the blood (conducted at the New York Hospital) showed a red cell count of 5,500,000, with 110 per cent hemoglobin; the carbon dioxide content of the venous and arterial system was definitely increased; the oxygen capacity and the viscosity were normal. On admission, vision of the left eye was equal to perception of light, and vision of the right eye was 6/200. There was slight exophthalmos of both eyes, together with divergence of the left eye. Examination of the fundus showed bilateral postneuritic atrophy of the optic nerve, with distended purplish veins bordered by a distinct broad whitish band along the perivascular space, due to a transudation of plasma; the perivascular space of the retinal arteries was not involved. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus associated with bilateral postneuritic atrophy of the optic nerve.

CASE 5.—A. B., a laborer 38 years old, was admitted to the medical ward of the New York Post-Graduate Medical School and Hospital on Jan. 10, 1935, complaining of severe headaches for one year and profuse diaphoresis. The past history was essentially unimportant. Examination showed definite cyanosis of the conjunctiva, lips and fingers. Examination of the capillaries of the nail beds of the fingers showed from two to three times the normal number in action, which is characteristic of polycythemia. Vision was normal in both eyes. The blood pressure was 130 systolic and 98 diastolic. The lungs were clear; the heart was normal; the spleen was not palpable. The red cell count of the blood was 7,800,000 with 142 per cent hemoglobin. The determination for the blood volume (per kilogram of body weight) totaled 104 cc. (the normal is from 82 to 88 cc.). The oxygen capacity of the blood was 29.3 volumes per cent (the normal is from 17.5 to 21.4 volumes per cent); the carbon dioxide content was 61.4 volumes per cent (the normal is from 48 to 64 volumes), and the oxygen content was 18.1 volumes per cent (the normal is 14.6 volumes per cent). Examination of the fundus showed distended purplish veins, edema of the disk and two small retinal hemorrhages in one eye. The general diagnosis was primary polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus, with edema of the disk.

CASE 6.—M. K., a boy $8\frac{1}{2}$ years old, was a patient in the clinic of the Gouverneur Hospital. Symptoms of heart disease had been present for five

years. There were marked cyanosis of the lips and tongue and clubbing of the finger-tips. The patient complained of dyspnea and fatigue on exertion. The spleen and liver were not palpable. The red cell count of the blood was 9,300,000, with 160 per cent hemoglobin; the white cell count was 10,000. The blood pressure was 80 systolic and 50 diastolic. Vision was normal in both eyes. Examination of the fundus showed distended, slightly tortuous, bluish retinal veins; moderately dilated, purplish retinal arteries; a reddish disk, and a fundus with a grayish tinge. The general diagnosis was congenital heart disease (pulmonary stenosis) associated with secondary or compensatory polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CASE 7.—F. D., a married man, 32 years old, was admitted to the clinic of the New York Post-Graduate Medical School and Hospital in 1929, complaining of vertigo. He had blueness of the face, lips and tongue. There had been shortness of breath for ten years, and clubbed fingers were noted. The blood pressure was 102 systolic and 76 diastolic. The spleen was not palpable. The red cell count of the blood was 9,790,000, with 130 per cent hemoglobin. The color index was 0.91. Vision and the field of vision were normal. Examination of the fundus showed distended, slightly tortuous, bluish retinal veins and moderately dilated, purplish retinal arteries. The physiologic cup was bluish; the color of the fundus was normal. The general diagnosis was congenital heart disease associated with secondary or compensatory polycythemia. The diagnosis of the condition of the fundus was fundus polycythemicus.

CONCLUSIONS

Cyanosis retinae is associated with many diseases. For this reason it is suggested that the term should not be limited exclusively to the condition associated with congenital heart disease but should be applied to the condition in any disease, including polycythemia, as this disease is accompanied by cyanosis.

Seven cases of polycythemia are reported.

In the five cases of polycythemia of the primary type the retinal veins were distended and purplish, and the retinal arteries were of normal caliber and color. Complications occurred in two cases. Vision was normal in all cases except one.

In the two cases of polycythemia of the secondary type the retinal veins were markedly distended and were bluish, while the retinal arteries were moderately distended and were purplish. There were no fundic complications, and vision was normal in both cases.

The differences in the fundus in the two types of polycythemia corresponded with the findings already reported in the literature.

Ophthalmoscopic examination in the early stages of the disease is an additional aid in the diagnosis of both types of polycythemia.

It is essential that a general physical examination be made in order to establish the diagnosis of fundus polycythemicus, as at times the vascular lesion of the fundus is not apparent.

Drs. Dan Witt, H. O. Mosenthal and Moses Deren made the general medical examination in the cases reported.

SHORT STUDIES ON THE HISTORY OF OPHTHALMOLOGY

IV. SIR CLIFFORD ALLBUTT, THE APOSTLE OF MEDICAL OPHTHALMOSCOPY

BURTON CHANCE, M.D.

PHILADELPHIA

On Feb. 22, 1925, there died in England that "spiritual aristocrat of Medicine," Sir Thomas Clifford Allbutt, Regius Professor of Physic of the University of Cambridge. Sir Clifford, although famous throughout the last fifty years of his life for his teaching in general medicine and long the leader of the medical profession in England, was also, it should be remembered, one of the most valuable contributors to ophthalmologic knowledge, one of the first to use the ophthalmoscope and to extend its use among general practitioners.

Clifford Allbutt, the only son of the Reverend Thomas Allbutt, was born at Dewsbury, near the city of York, on July 20, 1836. He had only a younger sister. When a small boy he became the child companion of an invalid lady, a cousin of his father, on an extended visit to her at Ventnor on the Isle of Wight. There he had some private instruction. His formal education began later in St. Peter's School in York, founded in the seventh century, at which the famous Alcuin was once master. He left St. Peter's school in 1855, and, having gained a classical scholarship, was admitted to Gonville and Caius College of the University of Cambridge, from which he was graduated with high honors in the first class of natural science established there.

"Gonville and Caius" is the college at which scientific and medical courses are held. This illustrious college was incorporated in 1557 by Dr. John Keys, whose name was latinized as Caius, one of the great physicians of the sixteenth century; he served King Edward VI, Queen Mary and Queen Elizabeth and was president of the Royal College of Physicians. Caius had studied at Padua, becoming a physician in the faculty in 1541, and for two years lived with Vesalius. He returned to England at the request of Henry VIII and lectured on anatomy to the surgeons of London for twenty years thereafter. He was not only a great naturalist, the first English anatomist, but a great physician, besides being a distinguished antiquary and classical scholar. He, like Linacre, of Oxford, was a disciple of the new learning brought from Italy. "Caius" has always been a physicians' college; Harvey, Glisson,

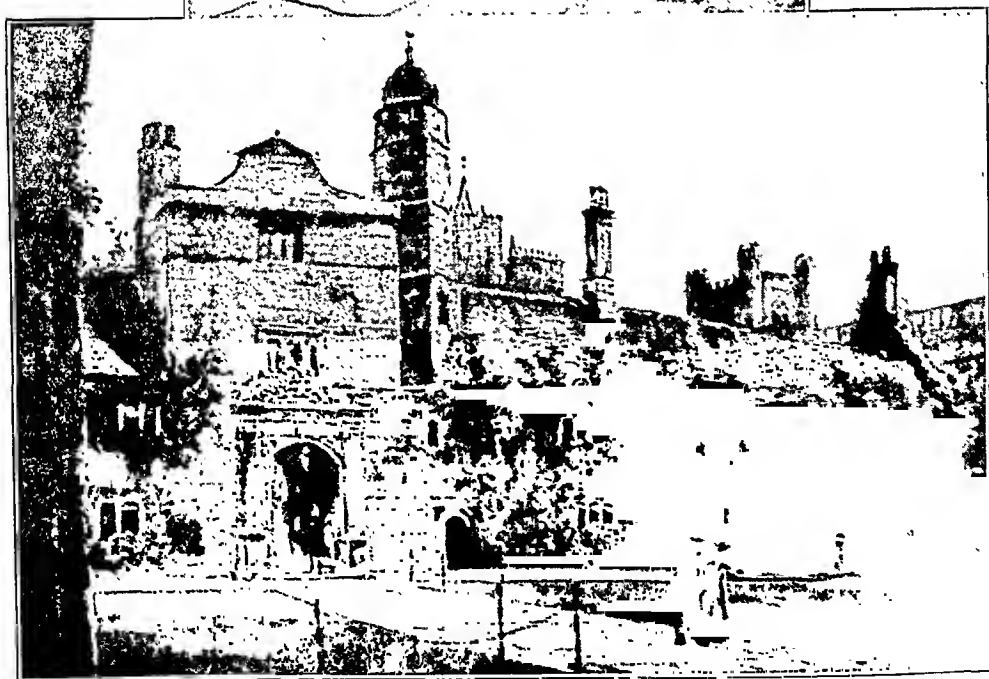


Fig. 1.—Two views of Gonville and Caius College, Cambridge University showing the Gate of Virtue.

the anatomist, and a long roll of eminent surgeons and physicians received their education there. Caius was master of the college of which he was the cofounder and generously contributed to its maintenance. The foundation stone he had himself inscribed in Latin, the equivalent being "John Caius dedicates this to Knowledge." He is buried in the chapel.

Dr. Caius, more than three hundred years in advance of the time, appreciated the importance of sun and air to the living organism by decreeing in the statutes of Caius "that no building shall be constructed which shall shut off the entire south side of the College lest for lack of



Joanes Caius

Fig. 2.—Portrait of Joanes Caius.

free ventilation the air shall become foul, and the health of the College impaired and disease and death ensue." Closed quadrangles had previously been built at all the colleges, but no more were built after the time of Caius. Allbutt must have been deeply impressed by the facts of the life of the founder of his college and must have been actuated to an emulation of Caius' activities.

When Allbutt entered Cambridge University his tastes and inclinations were more literary and artistic than scientific. Besides being a serious student of music, he became a pre-Raphaelite; later, when he visited Italy, he aspired to be an artist, but as he lacked the power of

expressing himself in painting he abandoned that desire. While at Caius his reading naturally widened, and he was impressed by the philosophy of Auguste Comte, so much that he decided on medicine as a career.

In his boyhood he was attracted to things medical by his visits to two uncles of his in the neighborhood who were physicians; they allowed the lad free access to their old-fashioned surgeries, where he learned the names and uses of the various drugs on their shelves. He had formed a museum of his own at one time, but all those interests were forgotten during his classical studies; they were revived, however, as has already been stated, by the contemplation of the discoveries which were then being made in biology, and through such influences he was led to begin the study of medicine seriously. Accordingly, he was entered at St. George's Hospital, London, where he had the good fortune to receive instruction from such illustrious teachers as Bence Jones, the pupil of Liebig, Sir George Paget and Sir Humphrey Murray. The inspiration exerted by these teachers endured throughout his lifetime and led him on to further study. By having taken the full curriculum and much extra research he was enabled to receive at Cambridge the degree of Bachelor of Medicine in 1861; but he did not receive his degree of Doctor of Medicine till 1869.

Following the advice of Bence Jones, whose clinical clerk he became, after receiving his baccalaureate Allbutt went to Paris, where, allying himself with Trousseau, of the faculty of the University of Paris, he began the study of the diseases of the heart and blood vessels, and, in association with Duchenne, began the observation of nervous diseases. Fascinated by certain aspects of dermatology, he attended the course given with great charm by Bazin. He returned to England by the end of the year and resumed his studies, especially under the direction of Sir William Jenner. Already he had shown evidences of the excellent tutoring that he had received and began to manifest an interest in pathology, especially the pathology of the nervous system, and through the guidance of Dr. John W. Ogle, the same teacher who had so greatly influenced Hughlings Jackson, he became seriously interested in a course in ophthalmology.

In 1861 Allbutt entered into practice in Leeds, in which city of woolen mills he was to reside for twenty-eight years. He attached himself to several institutions, among which were the House of Recovery, one of the early "fever-hospitals" in England, and the Leeds General Infirmary, to which he was appointed physician. His success was immediate. Two years later he received the appointment of lecturer on anatomy, comparative as well as human. He continued his association with the Leeds General Infirmary for twenty years and became one of a band of physicians remarkably distinguished in medi-

cine and surgery. Leeds was noted more for surgery than for internal medicine. Allbutt became allied especially with Mr. T. Pridgin Teale Jr., who later became a well known ophthalmic surgeon.

OPHTHALMOLOGIC INTERESTS

As has been stated already, Allbutt, while at St. George's Hospital, by his association with Dr. John W. Ogle, became interested in pathology, especially the pathology of the nervous system. During his conferences Allbutt received from Ogle the idea of the value of the ophthalmoscope in disease of the central nervous system. It is well to be reminded that it was due to his acumen that Ogle, soon after it had

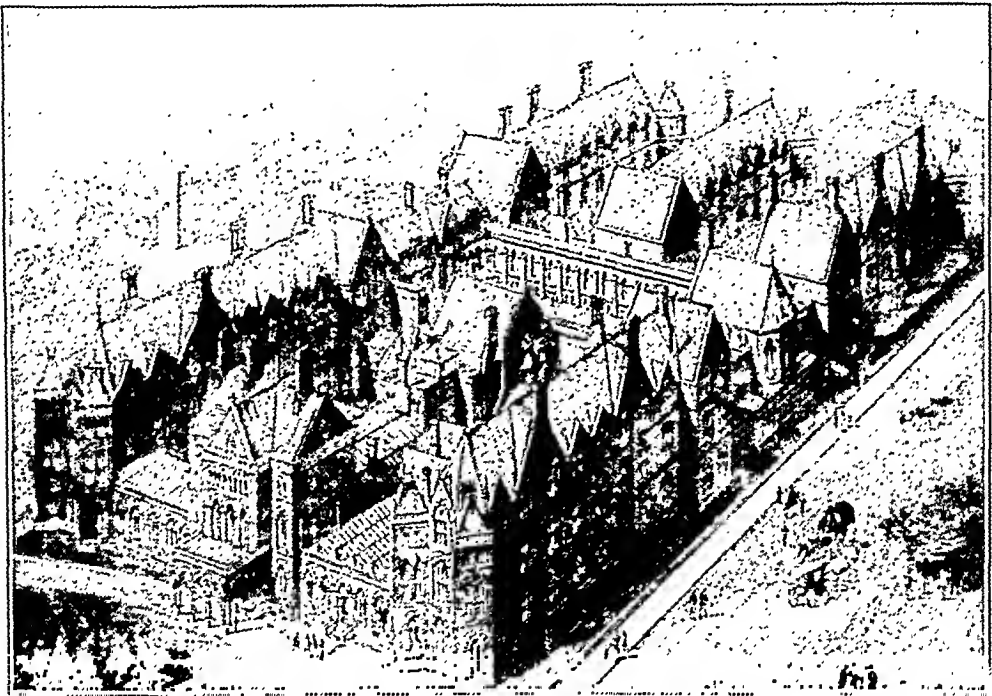


Fig. 3.—Leeds Infirmary, 1867.

been discovered, became convinced of the possible usefulness of the ophthalmoscope. In a paper which he published in 1860 Ogle¹ set forth the value of ophthalmoscopy in the study of disease of the brain and drew attention to "the very close relation which exists between the cerebral and the intraocular circulation." And he predicted that "the vascular structure of the posterior parts of the eye might serve in its variations as an index to the vascular conditions of the intracranial organs."

As Mr. Teale and Dr. Allbutt worked together at the infirmary on diseases of the eye, the former from the special side of the study and the latter from the more general aspect, they brought under observa-

1. Ogle, John W.: *M. Times & Gaz.* 1:572, 1860.

tion a great number of important cases in which the patients presented the ocular disease coincidental with other diseases, which otherwise would have been overlooked. Allbutt was aware that changes might exist within the eye along with disease of other organs, but as yet the laws of their occurrence were not known.

Mr. Teale had become expert in the use of the ophthalmoscope and, naturally, constantly employed it in his ophthalmic clinic. He had had forms made on which the fields of vision could be readily mapped, which maps taken at various periods of the same case he and Allbutt found often presented curious departures from what was at that day considered to be the normal.



Fig. 4.—Dr. John W. Ogle.

These two, colleagues and confrères, began an association which lasted for many years, both living in friendly intercourse well beyond their octogenarian days. It is to that fellowship that ophthalmology in the north of England owes its foundation in scientific principles and its establishment as an integral part of the practice of medicine. They early began in reporting cases from their joint practice to describe the changes within the eye found accompanying disease of other parts of the body, particularly of the central nervous system. Their articles included papers on locomotor ataxia, epilepsy in association with disease of the optic nerve, atrophy of the optic nerve dependent on orbital disease, retinal diseases in association with disease of the kidney and atrophy of the optic nerve following typhus.

Mr. Teale gave to Allbutt "of himself, his experience and his materials so generously and so continuously, that Allbutt could no

longer define between that which was his own and that which really belonged to Mr. Teale." In various other ways these two physicians were associated throughout their long lives until the death of Teale separated them in 1924. Sir Clifford, in a letter to me shortly after that event, remarked that his "dear old colleague Pridgin Teale" had recently passed away, aged over 90, "bright up to the last."

Among the friends of Allbutt in Leeds was Dr. James Crichton-Browne. Only a short time before Allbutt's advent this physician had been appointed medical director of the West Riding Hospital for



Fig. 5.—T. Pridgin Teale Jr., from a photograph taken about 1860, presented by his son, Mr. Michael A. Teale.

the Insane, located at Wakefield, about 10 miles (16.1 kilometers) from Leeds, containing about 1,500 beds. The enthusiastic director attracted there young men eager and keen like himself, and every facility for research was afforded them. In this band were David Ferrier, Hughlings Jackson, William Turner, Lauder Brunton, Milner Fothergill and others. Associated with them was Mr. Swanzy, then rising with distinction in ophthalmology. For years this band maintained their forces and pushed on together. Dr. Crichton-Browne, now Sir James, is still living, well beyond 97 years of age, enjoying an active intellectual life at Dumfries, on the east coast of Scotland.

It was not until several years after the invention of the ophthalmoscope that this instrument was much used. Mr. Spencer Wells,² in 1853, was among the first in England to insist on the great value of the ophthalmoscope in diseases of the eye. In their enthusiasm over the possibility of at least viewing the ocular fundus many who employed it were oversanguine as to the meaning of the appearances of the fundus in their relation to the brain. The ophthalmoscope was hailed as a sure means of making an accurate diagnosis as well as an advance in



Fig. 6.—Dr. James Crichton-Browne in early years.

the understanding of the pathologic features of general diseases, thus serving as an effectual aid in therapeutics. Its advocates saw that the instrument would make possible an improvement over the methods that were then available in the diagnosis of cerebral troubles and that it would provide the means to discover the retinitis dependent on renal and other remote disorders. After a while reaction set in, and expectations began to be disappointed. Physicians unskilled in the use of the instru-

2. Wells, Spencer: *M. Times & Gaz.* 7:264 (Sept. 19) 1853.

ment and unable to interpret the images revealed to them began to make blunders, so it was laid aside and, for a time, neglected. In the meantime a few patient workers toiled steadily in collecting data, comparing evidence and eliminating sources of error. Prominent among these workers was Clifford Allbutt, who, at the meetings of the various medical societies and in the columns of the *Lancet* and other journals, methodically explained the use of the ophthalmoscope, defined the range of its usefulness and described the phenomena made apparent by it.³



Fig. 7.—Spencer Wells.

After a year or so, however, Allbutt was gratified to learn that the ophthalmoscope was being more and more used by physicians. He was always careful to announce that it was Dr. John W. Ogle who had first drawn attention to the value of the instrument to physicians. The wide district from which the Leeds General Infirmary was supplied and the number of outpatients soon gave opportunities for such investiga-

3. Allbutt, Thomas Clifford, obituary, *Lancet* 1:461 (Feb. 28) 1925.

tion. In spite of this practice Allbutt declared in his monograph published eighteen years later that "the number of physicians who are working with the ophthalmoscope in England may be counted upon the fingers of one hand. Many practitioners shrunk from the use of the ophthalmoscope because they believed that a troublesome arrangement of light and darkness was a necessary condition." It would seem from this backwardness that ophthalmoscopy was looked on as a difficult procedure not easily carried out.

As a matter of routine Allbutt examined the eyes of his patients in the hospital, and he hoped that his method might soon be used by physicians generally. He sought to simplify his examinations and to make

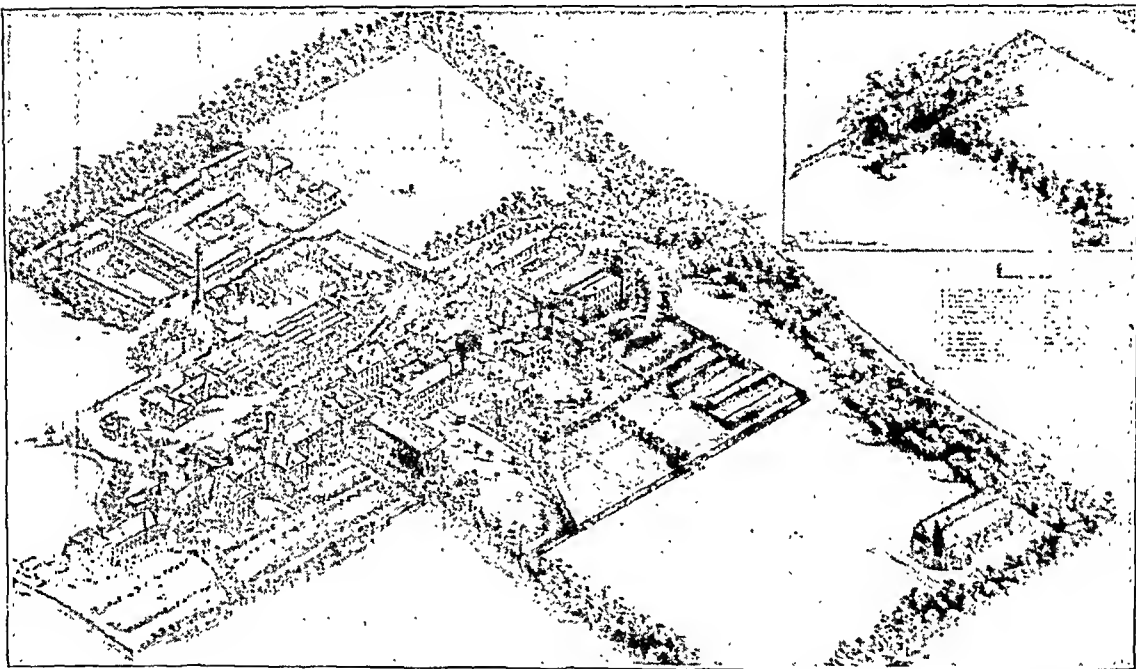


Fig. 8.—The West Riding Lunatic Asylum.

his explanations easy for beginners. He prepared diagrammatic outlines of the fundus on which could rapidly be sketched accurate representation on the state of any particular disk or retina. He endeavored to group together the patients with normal and healthy eyegrounds in order that his students might learn to define the natural details before passing on to other groups in which the picture was expressive of a departure from the normal and to groups with pathologic anomalies.

It was not long before Allbutt was able to arrange the details of his studies so systematically that they served as material for a course of lectures on "Optic Neuritis as a Symptom of Disease of the Brain and Spinal Cord." But as a preliminary to this formal presentation he recounted in much detail the analysis of his observations on a series of

several varieties of nervous diseases which he made in the examination of patients under Dr. Crichton-Browne's care. This was delivered under the title "The State of the Optic Nerves and Retina as Seen in the Insane" at a meeting of the Royal Medical and Chirurgical Society on Tuesday, Feb. 23, 1868.

While at Wakefield, Allbutt became greatly attracted to Hughlings Jackson, "whose simplicity, truthfulness and acuteness in the observation of disease and whose genuine insight into facts so long obscured by the cumbrous and vain phraseology of the Schools made Jackson's friendship as valuable to Allbutt as a student of nature, as the like qualities in his personal character won Allbutt's warm regard as his friend." Thus it was that Hughlings Jackson, in London, and Allbutt, in Leeds, began to use the ophthalmoscope in the investigation of cerebral disease at about the same time. That common interest procured for Allbutt the pleasure and the advantage of Jackson's friendship.

By 1867 Allbutt had accumulated from his experience sufficient material to publish a paper under the title "The Ophthalmoscope in the Physicians' Practice at the Leeds Infirmary."⁴ This paper created much comment and was followed the next year by "Optic Nerves and Retinas of the Insane,"⁵ "Medical Ophthalmology,"^{6a} lectures on "Optic Neuritis,"^{6b} "The Use of the Ophthalmoscope in Tubercular Meningitis and Spinal Disease"⁷ and "Optic Neuritis in Pyaemia."⁸

In the preparation of these papers was laid the foundation for a remarkable monograph which appeared four years later, "On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys; Also in Certain Other General Disorders." While this work dealt with the subjects of the earlier special papers, nothing was transferred from them into the book, the first issued in English on the subject of ophthalmoscopy. In this work Allbutt expressed his own conclusions based on the vast amount of material which he had been gathering. It is interesting to note the list of the subjects presented.

After the "Aspect, Structure, and Connections of the Normal Optic Nerve and Retina," are dealt with "Variations from Health" are described. Then Allbutt defined "The Relations Between Certain Intracranial Disorders and Affections of the Optic Nerve and Retina," which was followed by a description of the "Ophthalmoscopic Signs of Diseases of the Spine," and retinitis dependent on albuminuria, on leukemia and on syphilis, each exhaustively described. Following these

4. Allbutt, Thomas Clifford: *M. Times & Gaz.* 1:494, 1867.

5. Allbutt, Thomas Clifford: *Med. Chir. Soc. London* 51:97, 1868.

6. Allbutt, Thomas Clifford: *M. Times & Gaz.* (a) 1:495, 521, 574 and 628, 1868; (b) 2:61 and 116, 1868.

7. Allbutt, Thomas Clifford: *Lancet* 1:596, 1869; 1:76, 1870.

8. Allbutt, Thomas Clifford: *M. Times & Gaz.* 1:691, 1868.

are chapters on the amaurosis of diabetes and of oxaluria and on the toxic amauroses. An interesting chapter details the "Effects of Disorders of the Menstrual and Other Secretions upon the Nerve and Retina." The final chapter tells of "Embolism of the Central Artery of the Retina and Its Branches." In an appendix are recounted numerous histories describing the ophthalmoscopic changes which Allbutt had observed in cases of insanity in association with epilepsy, and of mania in association with idiocy and in association with "general paralysis" (*dementia paralytica*). In all these cases the author confined himself to those changes which he had looked on as having a symptomatic value, making no attempt to describe evidences of local disease.

In the preparation of his work on the ophthalmoscope, Allbutt was thoroughly assisted by Dr. Crichton-Browne, who aided him in many ways, especially in supplying and describing pathologic specimens, besides fully and unselfishly contributing to his undertakings. Allbutt had had unusual opportunities at the hospitals for patients with mental disorders in the other "Ridings" in Yorkshire. So constantly was he encouraged in his studies by Jackson that he dedicated the book to him.

In the meantime the great West Riding Hospital for the Insane at Wakefield was thrown open to Dr. Allbutt. It is difficult to conceive that a more magnificent field for the study of cerebral disease could be found. Every form of the diseases to be observed was continually represented there; as many as 1,200 patients were then under Dr. Crichton-Browne's care. That generous and enthusiastic superintendent permitted the students of the Leeds School of Medicine to avail themselves of those great advantages.

In the second half of 1867 Allbutt undertook the ophthalmoscopic study of the patients at the West Riding Hospital for the Insane under the direction of Dr. Crichton-Browne, and at the North and East Riding hospital for the insane at Clifton, near York, under Dr. Christie, and he was greatly aided by these physicians. The conditions had been diagnosed and the cases selected for him; many were observed repeatedly, even three times. So numerous were the patients and so varied their conditions that he could not refrain from regarding a hospital for persons with mental disorders as a "museum of cerebral disease." In some instances abnormal states of the nerves were found; in others, a normal state. For convenience the conditions were classified as "general paralysis" (*dementia paralytica*), idiocy, dementia, mania, monomania and melancholia. The cases of insanity depending on a connection with epilepsy were regarded separately.

Allbutt had been led to inquire into the phenomena of insanity from a general interest with cerebrospinal disease; his primary object was the hope and expectation of finding in the retinas of patients with dementia

paralytica changes of or about the blood vessels such as several observers had described in the brain in persons who had died of that disease. Although he was disappointed, yet he was led to the discovery of a certain condition of the optic nerve which will be described later, and he determined to continue his examinations with the hope of finding more definite knowledge of various pathologic conditions than was appreciated in the study of nervous disease at that time, relating to a sorting out of the organic from the functional changes. Also, he expected to find from the study of the eyegrounds in the course of mania some indication of the state of the cerebral parts of the eye as related to the state of the cerebral circulation.

Allbutt put no reliance in statements concerning the visual powers. He regarded the failure of sight as estimated by the ability to read test types as "an oculist's symptom"; the results bore but little proportion to the amount of the atrophic or other diseases seen in the nerve. The mapping of the field of vision, however, he declared to be "a physician's symptom" and to be of the utmost importance in some cases. Both these tests were out of the question with patients with mental disorders.

Ophthalmic surgeons then, as nowadays, were seldom called on to examine a nerve in the early stages of the neuritis, because at these stages there was not noticed marked loss of vision. In the incipient stages atrophy can seldom be surely ascertained; its chief significance, therefore, lies in the important pathologic changes which have taken place. Allbutt regarded this process as chronic optic neuritis. He found that atrophy bore no fixed proportion to the ataxia of the orbital muscles that one sees in cases of dementia paralytica, but usually the changes in the nerves were proportionate to the contraction and dilatation of the pupils. In the early hyperemic stage the pupils are contracted, but they become dilated as white atrophy succeeds.

He scheduled 53 cases of dementia paralytica, in which he found no changes in the optic nerve and retina. In 48 others he found atrophy of the optic disk in various stages, and 7 he marked as doubtful. He came to the following conclusions: In almost every case of dementia paralytica at about the end of the first stage there were ophthalmoscopic evidences of atrophy of the optic nerves, which he believed traveled slowly from the centers and along the optic tract. Later, after he had made a large number of sections of the tracts and the corpora quadrigemina, he was forced to reverse his conclusion. At first the disks were hyperemic, with a slight exudation on their surface and without much stasis. Later these signs became white and staring, the edges of the disks being sharply defined. In other cases the exudation might be decided and in the end appear as though punched out.

He noted 51 cases of mania. In 25 cases, a surprisingly large number, the state of the nerves denoted symptoms of intracranial

disease. In many others permanent changes in or near the disk were noted. In the cases of mania dependent on organic as well as functional causes, within a few days after a paroxysm the back of the eye exhibited vascular suffusion, so pink in certain cases as to obscure the disk. During the paroxysm, however, the disk was anemic, perhaps from spasm of the vessels. Exudation appeared only when the damage was permanent, as from obstruction to the intracranial circulation followed by atrophy. The frequency with which hyperemia of the disk and retina were observed after a paroxysm led Allbutt to conceive the idea that he could detect cases of mania by that appearance only. In certain cases the conjunctiva became hyperemic, owing doubtless to paralysis of the vasomotor nerves. He imagined that the anemia of the retina and the whitish state of the disk, a probable atrophy, might indicate that the eye participated in a disturbance in the circulation of the brain whereby a spasm of the arteries produced epilepsy of the mental functions followed by paralytic dilatation of variable duration.

There were 48 cases of dementia not connected with epilepsy, in most of which the condition was severe, being due to organic disease of the brain. In 23 he found disease of the optic nerve and retina.

In a large proportion of the cases of melancholia and monomania the condition was functional only. Among 17 patients, the retina and optic nerve were healthy in 10. One patient, who later became ataxic, presented signs of commencing atrophy of the disk. The ataxia confirmed Allbutt's opinion that the signs in the disk were in their early stage. He frequently noted the presence of anemia of the retina in patients with melancholia.

Forty-three cases of insanity depending on epilepsy were studied, but in comparison with the other classes the findings were few and of no distinctive importance. Epilepsy alone, that is, when it is not dependent on organic disease, was not usually accompanied by disease of the optic nerve. Organic disease, however, was indicated by signs in the optic nerve unilaterally. The class of patients with this condition, and all the other patients were given repeated examinations, and the diagnoses were confirmed by several independent observers.

Among the idiots, atrophy of the optic nerve was not uncommon. Among 12 patients, 5 presented decided atrophy, and another showed advancing disease. Whether this condition had been caused by encephalic inflammation in childhood Allbutt left for future observers to decide.

The main interests of Allbutt's communication lie in the schedules that he so perfectly prepared to show the details of his examination of 219 persons. In a large proportion of cases of old or severe organic disease of the brain and its membranes, whether degenerative, hemorrhagic, meningitic or due to tumor, changes in the eye were seen. In

the cases of so-called functional disease such changes were found in a few instances. At that early day of scientific neurology the occurrence of atrophy of the disk as that condition is now seen, as a constant symptom in cases of dementia paralytica, became a fact of the greatest pathologic importance. In cases of functional mania and functional epilepsy the variations of vascular tension in the retina and disk suggested to Allbutt and his colleagues fields of observation for later investigation.

Allbutt was persistent and most searching in his examinations and employed every available method and maneuver. He made repeated examinations in any state of the optic disks, pursuing his examination whenever possible, even during the course of an epileptic seizure or in coma or during a maniacal attack. His testimony settled for all time the fear of those timid physicians who refrained from using the ophthalmoscope lest the eye of the patient might be damaged by the heat and glare from the mirror. He did not fear to use a strong light, despite Jaeger's warning that strong lights might be dangerous.

Allbutt's studies led him to certain conclusions as to the value of the ophthalmoscopic signs in the diagnosis concerning cerebral diseases; he noted especially that in nearly every case of dementia paralytica atrophy of the optic nerves may be observed throughout the whole course. Allbutt was the first, in England at least, to make this observation. It is true that Westphal, of Berlin, with von Graefe's assistance, was similarly engaged; his researches were far less complete than Allbutt's, yet his conclusions were the same.⁹ These findings were not then considered to be of special diagnostic value, yet they were of great pathologic significance as throwing light on the minute changes of structure which cause the more characteristic symptoms of that disease.

These studies yielded the investigator much about which he had had no previous knowledge; his honesty compelled him to make inquiries as to what observers in other countries had seen and noted. When Allbutt began his studies he was ignorant of the German language; to further his investigation he learned German and was able to know what von Graefe and others were accomplishing in their labors in the same field. He therefore was greatly encouraged on learning the results of their independent records.

The lectures on optic neuritis formed part of a course in clinical medicine delivered at the Leeds School of Medicine. The lectures were necessarily not exhaustive, as they were designed for practitioners and senior students. Allbutt, after giving a short sketch of the anatomy of

9. Westphal, C. F. O., in Allbutt, C.: *On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys*, New York, Macmillan & Co., 1871, p. 193.

the parts of the eye with their connections and origin, described the vascular parts of the optic nerve and retina and their connection with the brain. Then he gave a short explanation of the ophthalmoscope, at that time employed by him by the indirect method which produced the reversed image of the back of the eye. After a brief account of the fundus, the details of which were obtained only after full mydriasis induced by atropine, the lecturer pointed out that the retina was not the expansion of the fibers of the optic nerve but rather a structure distinct from the optic nerve, possessing a separate system of nutrition. Despite the fact that they are antiquated, the lectures are of great interest even today and are as vital as when they were delivered. They show how the knowledge had developed from the first views ever obtained of the ocular fundus up to the days when the gross details were made visible by instruments vastly more efficient than Helmholtz' original invention and had become so acceptable in their definition as to justify the preparation of an atlas, such as Liebreich's, to serve as a guide for all observers of the fundus. By this time it had become apparent that the eye was an outpost of the brain. Though the minute details of the retina and optic nerve had not yet been seen, sufficient was known of the apparent small details to justify the assumption that the morbid changes seen were not confined to the ocular structures but were an extension of what was presumed to be existing within the cranium, made visible by the ophthalmoscopic mirror, so that the changes of "optic neuritis" could be described in detail.

It might be of interest to those who today are teachers in medical schools to read the advertisement which was put out by the Leeds School of Medicine. In the announcement for the winter session, to commence on Thursday, Oct. 1, 1868, Dr. Allbutt is scheduled for demonstrations in clinical medicine and for a course in the "Principles and Practice of Physic." And for the summer session of 1869, commencing on May 1, he is listed as giving a course in comparative anatomy. The clinical lectures were given at the Leeds General Infirmary. The fees for attending the course in medical practice were, for the winter session, £ 7, 7s.; for the summer session, £ 6, 6s.; for twelve months, £ 12, 12s.; for eighteen months, £ 15, 15s.; for three years, £ 21, and in perpetuity, £26, 5s.

In January 1868 Allbutt¹⁰ presented an analytic and critical review of a number of papers by various authors published in England and France from 1860 to 1867 on subjects connected with the ophthalmoscopic study of alterations of the optic nerve and of cerebral diseases on which such alterations depend. At that time knowledge of the

10. Allbutt, Thomas Clifford: *Brit. & For. Med.-Chir. Rev.* 41:127 (Jan.) 1868.

diseases of the cerebral nervous system was meager, largely because of the lack of method in their investigation. The functions of the brain and the complexity of the phenomena as they were then understood gave rise to theories incapable of being tested by experiment and unchecked by reference to nature. The application of the ophthalmoscope, an instrument devised for the study of the eye and its particular structures, to the diagnosis of nervous disorders, however, gave one an insight into the necessity for the utilization of other instruments of precision which at that time were crude and inefficient. Already, Ogle, Allbutt and Jackson, and Carter and Hutchinson, in England; Galezowski and Bouchut, in France, and von Graefe and Saemisch, and Schweigger and Sichel, in Germany, were using the ophthalmoscope and appreciating the marvelous changes which the instrument had produced in the knowledge and method of the oculist.

In the review Allbutt presented a systematic yet brief description of the ophthalmoscope, the methods of using it and the details of the ocular fundus, with the straightforward simplicity and clearness which characterized all his writings. In this account were revealed the signs of the diseases of the optic nerve and retina which he observed in the analysis of the cases that he had studied at Wakefield hospital for the insane. His article must have made a strong appeal to the cultured general practitioners of medicine and surgery familiar with the English "Quarterlies," who might not have been attracted to the occasional report in the weekly professional journal.

The essayist unhesitatingly declared that not only did the ophthalmoscope clear up many doubts and enable one to recognize certain pathologic states which before were beyond reach, but it encouraged new habits of accuracy, which habits became reflected in the work of other departments of practice where the ophthalmoscope was less needed. The ophthalmoscope helped the science and practice of medicine not only by the facts which it directly revealed but stimulated work in the direction of nervous diseases especially, for by means of the ophthalmoscope one was enabled to see for the first time in the history of medicine the commencement and progress of changes in the life of nervous tissue and to ascertain the modes and times of such changes. Through the teaching of those whose works were referred to it became clear that the instrument should have been in the hands of every physician who wished to speak with authority on the subject of diseases of the nervous system.

In the perusal of these pages written nearly seventy years ago one must bear in mind that at that day the ophthalmic practitioner was not accorded the place in the estimation of the general practitioner which was ascribed to him twenty years later. The review said just enough to show that various and important indications of disease were to be

found in the eye and that physicians were in possession of a vast number of facts which helped in the diagnosis of many hitherto obscure diseases and marked states of tissue.

By this time Liebreich's atlas of the fundus had been published. This contained admirable plates to show the chief diseases of the retina and optic nerve. So important were the facts now available that it became clear that they no longer could be neglected on the score of their minuteness and that instruction in ophthalmic practice should be given to both medical and surgical students. The review closed with quotations from Sir Thomas Watson, who through the influence of Allbutt's persistence began his course of lectures in medicine with four lectures on diseases of the eye and who declared that "we find in the eye more satisfactory and plain illustrations of the general facts and doctrines of pathology than in any other part of the body." And, to use the words of Dr. Latham, "Here you see almost all diseases in miniature, and you learn many of the little wonderful details in nature of the marked processes which but for the observation of them in the eye could not have been known at all."

Looking back at the events of 1865 to 1875, one knows that the words of Sir Thomas Watson and Dr. Latham daily gained weight and a meaning which previously could be no more than guessed at by their distinguished authors. The fruits of those workers settled for all time the truth of the assumption that damage or a change of state in the eye is not uncommonly coincident with nervous and other diseases. Even at this early day in his career Allbutt's influence became of prodigious importance, and as physicians became aware of the facts which he pointed out as necessary to be observed wherein he sought to strengthen inference into certainty they worked on. Yet they could attain no thorough knowledge of the significance of optic changes in association with cerebral disease until they had become familiar with the modes of the production of those changes. Between those concurrent events there lay a chain of causation which turned out to be a series of phenomena of infinite value, as amply attested by many cases of high interest.

In commenting on Dr. Hughlings Jackson's aphorism that "optic neuritis depends upon coarse intracranial disease," Allbutt stated the belief that such a doctrine does not apply to "chronic neuritis," and he inquired as to the value of that aphorism, not because he doubted the truth of Jackson's statement but because he wished to know what that scrupulous and thoughtful observer implied therein. Allbutt ventured to say that "Dr. Jackson was inclined to use words 'loosely.'" However, he did not imply that there was looseness in the thought but rather that, owing to the deficiencies in the contemporary knowledge, Jackson

was compelled to coin his own phrases for the meaning of which he alone became responsible among those who dare to employ such phrases. Allbutt declared: "They like loose words, therefore, not that they may easily slip them on, but that they may easily slip them off, so that there may be room for their thoughts to move at ease within them. Unluckily, however, there are many other people who gladly catch at any expressions that seem at once fine and easy." For this reason Allbutt was disposed to deprecate the use of such an aphorism as that of Dr. Jackson about optic neuritis and coarse disease, just as he deprecated the latter's use of the term "cerebral fever," which term fits too many conditions too easily and encourages in others the very mental indolence prevented in Jackson. In the series of 200 cases which Allbutt presented before the Royal Medical and Chirurgical Society he sought to show that optic changes were chiefly or entirely coincident with static changes in the brain centers, yet he hesitated to speak of those changes as indicative of "coarse" disease. However, he would call a tumor pressing on the cavernous sinus or severing the connection between the optic nerves and their ganglions coarse disease, but would not so denominate the belt of cerebritis around it. Dr. Jackson was inclined to regard a small abscess as coarse disease, but Allbutt would not consider a small spot of softening coarse. A rude clot may be far coarser than a small patch of meningitis about the chiasma, but the clot leaves the eye uninjured, while the meningitis sets up optic neuritis. Dr. Jackson would hesitate to say, "The coarser the disease the more the neuritis," and yet in Allbutt's opinion it should be so if the coarseness were a direct element in its causation. A clot, he would say, though coarse disease is not itself an encroaching disease; so he would propose a change in his friend's rule by saying that optic neuritis depends on encroaching disease, whether coarse or not, including not only ischemia and acute neuritis, but chronic neuritis also.

In the series of lectures Allbutt confined himself to the most obvious details and refrained from debatable subjects. Through all his lectures and at their conclusion he testified to the thoroughness and the attractiveness of the able papers of Hughlings Jackson.

The effects of his entire course of study of the cases referred to in these pages only encouraged Allbutt to maintain what he and Hughlings Jackson declared from the beginning of their careers, that the "account of a cerebral disease cannot be complete without the record of ophthalmic observations."

To serve as a general doctrine concerning optic neuritis, he ventured to pronounce certain aphorisms, such as: "Ischaemia is always due to pressure; Neuritis to encephalitis, cerebral or meningitic." In cases of tumor in which there were ischemia and neuritis also, Allbutt was not always able to satisfy himself as to the etiology, because the pathologic

features were not yet definitely understood at that date, although he was inclined to regard the tumors as ischemic without disease of the optic nerve trunks. Few writers in that decade used the ophthalmoscope in cases of cerebral disease. Allbutt determinedly sought for autopsies and performed them whenever possible; in numerous cases he observed inflammatory changes in the brain substance surrounding the neoplastic or cystic body and on the meningeal coverings. He became aware that such augmentations in the cerebral contents excited an increase in the intracranial pressure, which extended its force into the foramina in the basal fossae. Many physicians at that date held that ophthalmoscopic research could not reveal anything of the nature of a cerebral tumor; nevertheless, Allbutt sought every opportunity to examine and reexamine repeatedly the fundi of those suspected of having intracranial disease. He regarded the occurrence of optic changes as not only the one symptom among many but often the one important symptom among a few. He did not hesitate to decide on the approximate location of a tumor but stated that tumors of the base of the brain commonly implicated the optic tract, and, if present in the anterior fossa, they eventually would press on the cavernous sinus, the chiasma or the tract; tumors of the posterior fossa seldom interfered with the optic disks, but, when very large, they might have compressed the medulla and extended to the quadrigeminal bodies or were propagated to the cerebellar peduncle. Tumors of the cerebral lobes, he found, affected the optic nerves variously. Allbutt was compelled to rely on his own experience in presenting the accounts of their effects; he believed that optic signs sooner or later appeared in almost all cases of tumor in the anterior lobes and in the middle fossa, yet he had found complete bilateral atrophy in cases of tumor of the posterior lobes.

During his observations Allbutt was impressed by the truth of the fact which Hughlings Jackson had repeatedly remarked and which was insisted on by von Graefe, that although the optic disk and its neighborhood might be greatly changed central vision can still be normal. Another clinical item was to the effect that greatly swollen disks can become reduced and the resulting vision higher than that in cases of descending neuritis, in which latter condition the visible contour of the disk might be but little changed.

Again, Allbutt, like Jackson, regarded all statements as to visual powers as useless; in all instances he endeavored to map the visual field. The notes of his experience tell of the subjective and objective symptoms following involvement of the sections within the various fossae of the base of the cerebrum. He was well aware that disease of the

cerebellum resulting from pressure and from inflammatory change may cause amaurosis because of the nearness of the quadrigeminal bodies. Galezowski and other continental observers were inclined to declare for inflammatory processes, but Allbutt maintained that in the greater number of cases the condition was caused by tumor. Hemorrhage into the optic centers was invariably followed by amaurosis, though the effects on the nerve were not always shown early by ophthalmoscopic signs. The keenness of Allbutt's interest in the subject was manifested in the thoroughness with which he pursued his study of cases. He watched the results of all the cerebellar tumors in the patients under his care and noted their relations to the quadrigemini. He examined microscopically the tissues around the tumor, as well as the peduncles and the quadrigeminal bodies themselves. He remarked that acute diseases, such as cerebral hemorrhages, when occurring alone never caused ischemia or neuritis. A clot, as a rule, did not enlarge and arrest the movements or force of the blood but became contracted and consequently withdrawn into itself. Allbutt's belief was that "it is not a sudden thrust but a gradual invasion which does mischief." Yet clots were often antecedents or sequences of softening and were often associated with neuritis, generally with chronic neuritis or simple atrophy. Allbutt agreed with that most acute and trustworthy observer, von Graefe, but differed from Galezowski and others who rarely observed optic signs in cases of cerebral hemorrhage, in noting that hemorrhage in the region of the thalamus might exert pressure on the tract, with resulting hemiopia.

Allbutt had opportunities to observe from its commencement the process known at that early ophthalmoscopic day as "simple white atrophy," a condition observed by the ophthalmic surgeon then, as today, generally only when loss of sight had set in and the atrophic process was almost over.

As a name for certain lesser degrees of resistance to destruction which often precedes white atrophy Allbutt introduced the term "red softening of the optic nerve," by which he meant the presence of only slight congestive appearances, accompanied with but little disturbance of the cerebral vessels. When patients with the early stage of cerebrospinal disease were examined he usually found many presenting the signs of so-called simple progressive atrophy, which showed a distinctly hyperemic stage, with little effusion preceding the stage of whitening. This condition was not noted in the textbooks on diseases of the eye of his day, but to Allbutt's great delight he found that it had been

described in 1864 by Jonathan Hutchinson.¹¹ In a microscopic study of a case he found excessive fibrillation but no marked proliferation of the nuclei. Allbutt was not able to determine the pathologic cause of this condition; he found chronic optic neuritis in cases of dementia paralytica and locomotor ataxia. Allbutt had had many opportunities for postmortem examinations, an unusual privilege at that time in England. He observed frequently in advanced cases of dementia paralytica alterations in the optic papillae and nerves, in the tracts and in the quadrigeminal bodies, although in recent cases the corpora quadrigemina were not deeply affected. He did not find such changes in the cases of ataxia, however. At that time to Allbutt and to all others working on the subject the cause of chronic neuritis was obscure; certainly it could not be explained by Hughlings Jackson's idea that "optic neuritis depends upon coarse disease." Allbutt ascribed it to "encroaching disease whether coarse or not." When white atrophy was seen for the first time he did not attempt to ascribe it to any particular precedent process, nor did he try to distinguish the acute from the more degenerative form of neuritis. It was generally noticed that the two eyes were unequally affected, a fact which offered some help in the diagnosis. Complete atrophy was found to be present a year or two before decided symptoms of disease of the brain, such as paralysis, betrayed the real seat of the disease. In his teaching Allbutt insisted that the early symptoms should always be noted. The pupillary actions might vary; indeed, not always were the pupils dilated or even inactive. But the field of vision was always lessened laterally before central vision was much disturbed. Occasionally he found only one eye atrophic, and in some cases of blindness the disks presented no visible changes.

The monograph entitled "On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys; Also in Certain Other General Disorders," which Allbutt published in 1871, received general attention, as it well deserved, not only because it was the work of a highly intelligent and eminently scientific physician but because Allbutt had devoted his great abilities to the elucidation of what at that time was a literally dark subject. The use of the ophthalmoscope in ordinary medicine had been so recently adopted that only those physicians who had striven to use the ophthalmoscope systematically in their ordinary work could tell how difficult it had been to put the right interpretation on what they had observed. There was no book to which they could refer to aid them in their difficulties. The book was especially

11. Hutchinson, Jonathan: *Clinical Data Respecting Cerebral Amaurosis*, London Hosp. Rep. 1:33, 1864.

welcome to such physicians, while many to whom the ophthalmoscope had been an unknown instrument were encouraged to take it up and work with it since its value had been made clear to them.

Allbutt began with an account of the introduction of the ophthalmoscope into ophthalmic practice and its gradual application to the

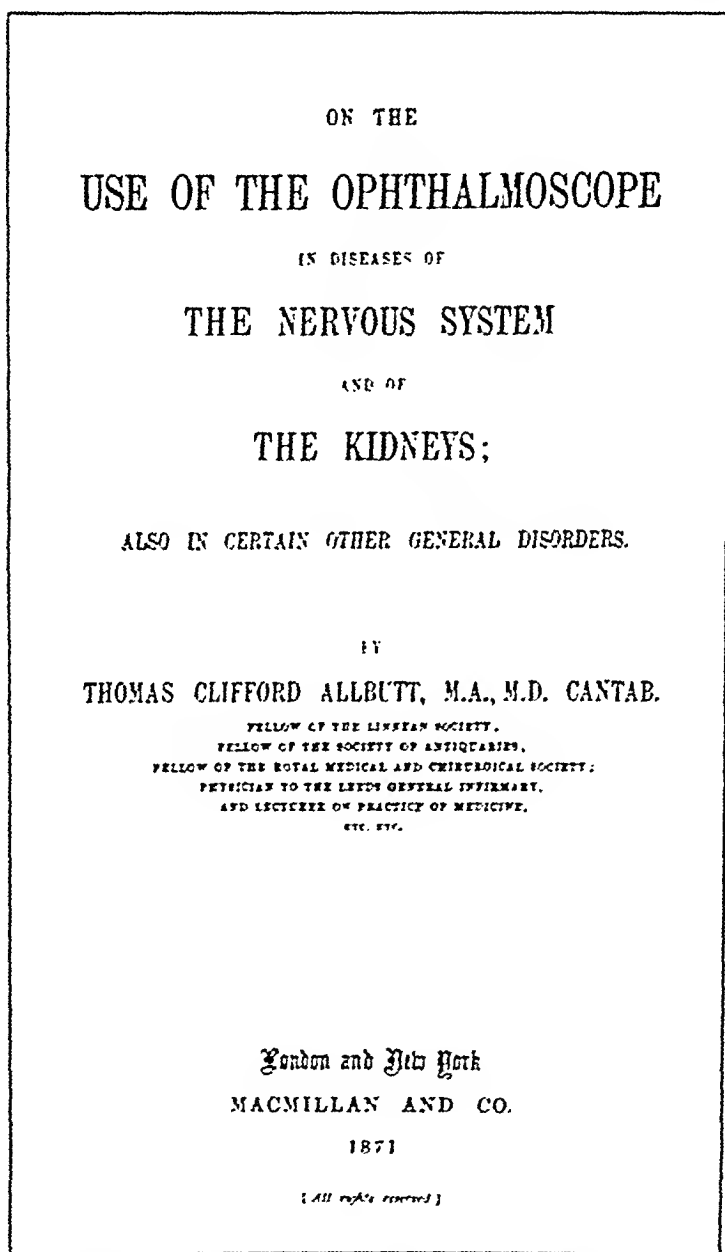


Fig. 9.—Title page of Allbutt's monograph.

elucidation of other than purely ophthalmic problems. Next he detailed the mode of using the instrument. For general use he employed a metallic mirror and two object glasses. He condemned all fixed apparatus as useless and cumbersome. He advised physicians "while learning to handle the apparatus to operate on an eye whose pupil had been

dilated by atropine; yet the sooner dilatation could be dispensed with the better, for patients did not like it, as the disordered accommodation remained long after the necessary examination, and normal vision was consequently interfered with." He assured all that they "might speedily get accustomed to dispense with the use of atropine." The succeeding chapter is devoted to anatomic details, the most important of which at that time seemed to have been that the vascular supply of the optic disks was connected with the brain system rather than with the retina and that the coverings of the optic nerve consisted in an actual prolongation of the arachnoid interspaces.

The anomalies of the optic disk, which are puzzling to the beginner, were next considered. Those alluded to were the varied distribution of the retinal vessels, persistence of the hyaloid artery, pulsation of the veins of the retina, the physiologic excavation of the disk similar to that existing in glaucoma, and the troublesome anomalies in the color of the disk, having an important bearing on the significance of hyperemia and anemia, as only the experienced observer could say what was physiologic or natural and what was pathologic or the result of change due to disease. Edema and ischemia were pointed out as being equally annoying, for the latter could be confounded with inflammation, commonly under the perplexing term optic neuritis. To differentiate the truly inflammatory process the term "neuroretinitis" was introduced, while the term "ischaemia" was applied to conditions superficially somewhat similar but due to vascular arrest.

The term "chronic optic neuritis" was retained to indicate the subacute inflammation which so commonly preceded white atrophy. "Retinitis" and "perineuritis" were pointed out to be of great importance medically, as in daily practice the diagnosis between "consecutive atrophy" and "primary atrophy" is not always easy.

The relations of these conditions, so lucidly described, to intracranial disorders were next considered, especially those cranial disorders involved in epilepsy, chorea, mania, dementia, meningitis, concussions and fracture, hydrocephalus, tumors and periostitis chronica, atheroma, softening and hemorrhage of the brain, cerebritis, abscess and sclerosis and, finally, dementia paralytica. Allbutt had noted that during the seizures of epilepsy the disk was somewhat vascular, the vessels being generally larger and fuller than usual, while in chorea no change was seen, as a rule. In acute mania, just after a paroxysm there was slight hyperemia, or pinkness. Many changes, but none very marked, were seen in dementia. The ophthalmoscope enables one to come to an early diagnosis in cases of the tuberculous form of meningitis. Long before other symptoms are visible, congestion of the optic papillae may give

warning of the approaching danger. Ischemia could be seen in the early stage of hydrocephalus, to be followed later by simple white atrophy. Much had been accomplished by the ophthalmoscope in connection with intracranial tumors. In dementia paralytica of persons with mental disorders observers had detected a tendency to simple white atrophy of the disks. Disease and injuries of the spinal cord often alter the optic disk, thus manifesting a connection between the two, but in 1870 much remained to be done before the ophthalmoscope could be relied on in the discrimination. The morbid conditions of the eye accompanying albuminuria, then spoken of as "albuminuric retinitis," afforded an opportunity for the ophthalmoscopist to draw valuable conclusions as to the existence or nonexistence of that malady. As the disease may exist in a marked degree without the appearance of dropsy, that fact is of sufficient importance to impel physicians to cultivate the use of the ophthalmoscope. And such might be the case in leukemic retinitis, with its peculiar and characteristic symptoms. Toxic amaurosis was touched on, and the classic story of embolism of the arteria centralis was mentioned. The book made no pretense to present a complete history of the morbid conditions of the retina in all kinds of disease; at that time no such compilation existed. The work served as an impartial review of the literature and was at once recognized as "full of sound reasoning based on honest observation, never carrying conclusions further than the available data justified, and assembling all the known facts about the subject" and "not entertaining," as the author quoted from Locke's 'Human Understanding,' "any proposition with greater assurance than the proof well warrants."

Dr. Ogle,¹² who must have taken great pride in having directed Allbutt to take up ophthalmoscopy, wrote as follows of the monograph on the ophthalmoscope:

The great merit of the work is, that it clears the way for other workers. By its aid men will no longer be compelled to work for years in the dark—they will have a definite standpoint whence to proceed on their course of observation. And we think it well to say a word on the spirit of the book, which is to us most pleasing. It is not a book of controversy; in it all fellow-workers are spoken of in the same loving spirit. From the kindly dedication to Dr. Hughlings Jackson to its "Finis," this treatise has only words of encouragement to those workers for whom the author lays bare all his stores—the accumulations of much hard work both in the field of observation and in that of research. Did it not seem unkind, we should be tempted to desire that a new edition might be deferred until these stores had fructified and produced the ample harvest of exact knowledge the labour spent in their acquisition deserves.

This important piece of scientific and literary work is exceptional in that, contrary to Allbutt's usual practice in later years, he never

12. Ogle, John W.: *Brit. & For. Med.-Chir. Rev.* 49:427, 1872.

brought out a second edition or returned at length to the subject. But he had hoped to follow up the volume by another essay on the disturbances of motion and nutrition of the eyeball. In a personal note to me written in November 1924, Sir Clifford, in response to an inquiry that I had made of him, referred to his monograph on the ophthalmoscope as "sere and yellow."

It is interesting to know that Allbutt for a time used the long tube ophthalmoscope of Lionel Beale, carrying the mirror, the lens and the lamp, such as that used by Hughlings Jackson. But he abandoned it because he "did not like to change the lenses at will, or to pass from the indirect to the direct examination, all of which is impossible in fixed instruments." He seems to have preferred a metal concave mirror of about 10 or 12 inches (25.4 or 30.5 cm.) focus, with a clip behind it to hold ocular lenses, much like the type devised by Brudenell Carter, with two convex object glasses, and a concave glass for the direct examinations. Much of this study was carried on by the indirect method; occasionally he employed a binocular or a stereoscopic instrument.

Notwithstanding the advice of Hughlings Jackson that he should work at medical ophthalmology, Allbutt did not enter ophthalmic practice but used his ophthalmologic knowledge to aid him in his general interest in clinical medicine. The disorders of the circulation which Trousseau had led him to observe particularly attracted and maintained his attention for many years, indeed, as long as he lived.

After comparative silence for a number of months Allbutt¹³ published a decidedly critical paper on the cause and significance of choked disk in intracranial disease. It must have been gratifying to him to see the fruits of his labors and to note that practitioners in England, France and Germany could not dispense with the use of the ophthalmoscope in the study of nervous disease. The headaches complained of by a syphilitic patient could not be understood nor the "biliousness" manifested in the course of an intracranial tumor explained without an ophthalmoscopic examination. In the case of syphilis the optic nerve might be inflamed or atrophic, while the effects of tumor might lead to congestion of the papilla, producing the *Stauungpapille* of von Graefe, or the "choked disc" of Allbutt. Through a natural increase in his knowledge since the preparation of his earlier papers and the monograph the author sat in judgment on his own contribution and that of other observers on the continent. He made a sharp distinction between optic neuritis, implying a true inflammation, descending through the course of the nerve, and choked disk. In the ten years of his experience he frequently encountered cases in which the elements were mixed, the diagnosis becoming therefore difficult to decide.

13. Allbutt, Thomas Clifford: On the Causation and Significance of the Choked Disc in Intracranial Disease, Brit. M. J. 1:443, 1872.

Few investigations in medical science and practice in the decade of 1865-1875 excited more interest than those made into the connection of the changes in the eye in association with diseases of the central nervous system. The various hypotheses advanced to account for the causation of choked disk both clarified and confused the pathologic conceptions held by all observers. Allbutt endeavored to present the

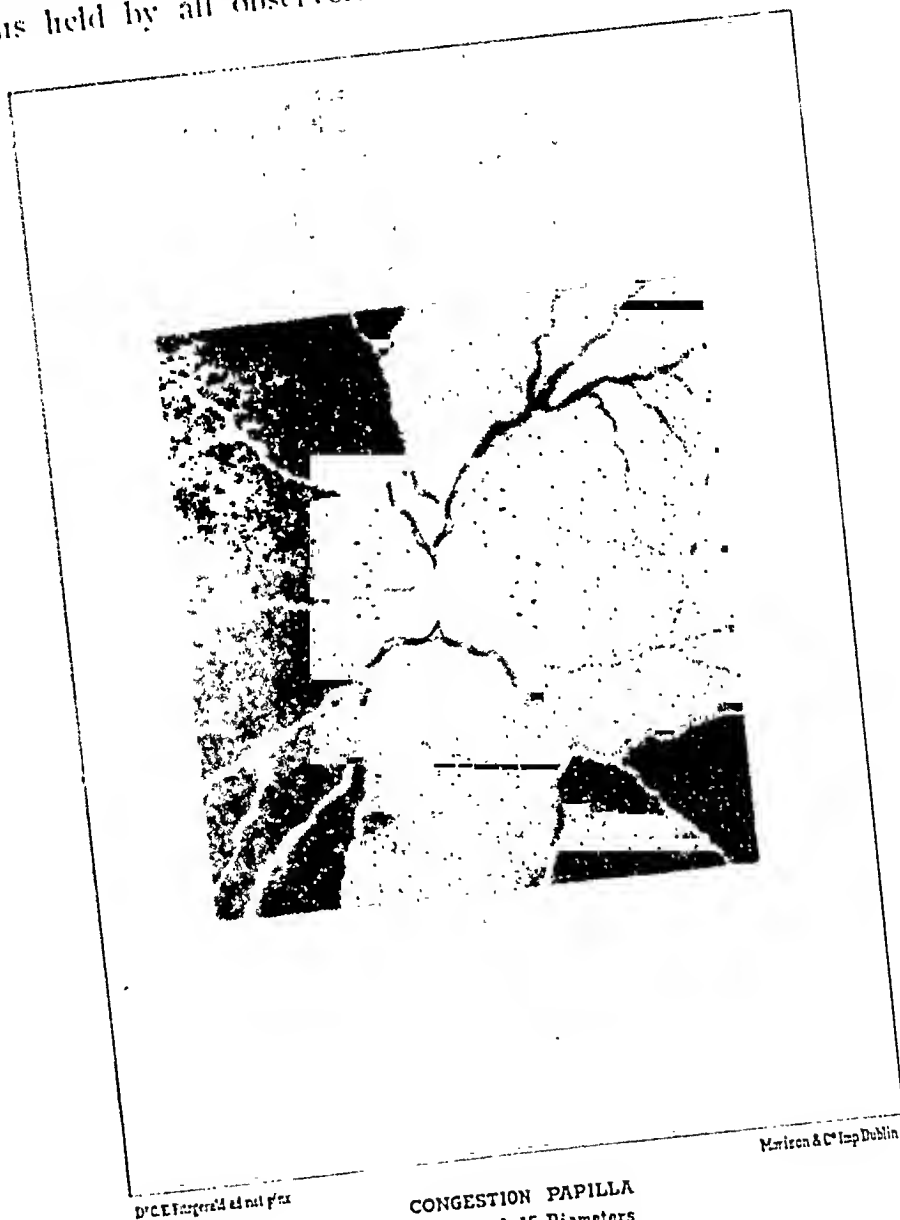


Fig. 10.—Frontispiece of Allbutt's monograph.

latest developments of the problems in his monograph. In his later and final paper on ophthalmologic subjects he referred to the investigations of Manz, Schwalbe, Schmidt, Axel Key and others. His theory that *Stauungspapille*, or choked disk, depends on excessive intracranial pressure coincided with that held by von Graefe. At that time he was

uncertain as to the mode in which the pressure makes itself felt. In effect he strongly believed that the intracranial pressure forced the fluids into the neural sheaths and that, owing to dropsy, the vaginal spaces pressed on the nerve and caused the swelling, the choking of the disk arising from the retention of the fluid in the nerve head by the narrow scleral ring. There seems to have been in most cases a definite relation between the amount of intracranial fluid and the dropsy of the nerve. As to the association of descending neuroretinitis and choked disk, Allbutt stated that he did not remember ever seeing a well marked swollen disk pass into the typical form of neuroretinitis. In conclusion, he had found no hypothesis which fully demonstrated the causation of descending neuritis.

In addition to reporting his own investigations in his chosen subjects, Allbutt occasionally wrote reviews of books and biographic notices of distinguished persons. There were two brief articles of interest to the ophthalmologist. The first is a notice of Galezowski's¹⁴ article on the diagnosis of diseases of the eye by means of retinal chromatascopy. Galezowski, who for years had been interested in color blindness, a condition which was for a long time in France cruelly spoken of as *daltonisme*, pointed out in his book that color blindness might be acquired as the result of certain conditions of the optic disk and retina and that colors might be employed in the tests for ascertaining the condition of the optic nerves. The second article of ophthalmologic interest was a sympathetic and generous appreciation of the life and works of Jules Sichel, who died toward the end of 1868. When one knows the characteristics of Allbutt one can see how readily he would appreciate the personal greatness of Sichel, who did so much to place diseases of the eyes on a scientific basis.¹⁵

For a number of years Allbutt occupied the chair of "Principles and Practice of Physic" and of "Materia Medica and Therapeutics" in the Leeds School of Medicine and was in charge of the museum, a fitting reminder of his boyhood interests. When he was made consulting physician to the Leeds General Infirmary in 1884, he resigned most of his lectureships but continued to be actively interested in the medical school. As president of the council he effected notable reforms over which he had control, in the system of medical education, and in time the medical school became a part of the University of Leeds. In his clinical lectures he adopted the methods of Trousseau, whom he much resembled. In his interpretations of the ideas of others he manifested

14. Allbutt, Thomas Clifford: Du diagnostic des maladies des yeux par la chromatascopie rétinienne précédé d'une étude sur les lois physiologiques des couleurs, by X. Galezowski (book review), *M. Times & Gaz.* **24**:490 (Oct.) 1868.

15. Allbutt, T. C.: *M. Times & Gaz.* **2**:676, 1868.

in a charming manner a singular clarity of exposition, which made a strong appeal to all the students, thus increasing his popularity with them.

He sought to lay practical stress on the true method of investigating disease. He endeavored to discover the minute and early deviations from health rather than to be concerned only with great examples of damage. He considered it one's duty to familiarize oneself with the general laws and modes of the growth of tissue as seen grossly or microscopically and also to learn the special modes of the growth of tissue for each particular part.

By his invention, in 1867, of a short graduated thermometer tube which could be employed readily on the person, the use of which became established as a routine procedure, Allbutt should be given credit for the introduction of thermometry into clinical medicine. One of the fruits of his interest in thermometry was a paper on "The Effects of Exercise on the Bodily Temperature," which he read before the Royal Society in the same year that his work on the ophthalmoscope appeared. And another paper, on the significance of cutaneous diseases in the classification of diseases,¹⁶ was published in 1867. In the next year, he published in *St. George's Hospital Report*, a short-lived periodical, an epoch-making paper of much importance to the ophthalmologist on the histologic changes in syphilitic disease of the cerebral arteries.¹⁷ It commonly has been asserted that Huebner was the first to make the observations reported, yet as Huebner's report was not published till 1874 the credit should be given to Allbutt for the discovery of this condition of the brain.

Disorders of the circulation particularly attracted Allbutt's attention. He introduced the terms "senile plethora" and "hyperpiesia" to describe the rise of the arterial blood pressure that occurs in otherwise healthy persons after middle life, especially noting the rise of tension in men who had discontinued athletic habits but continued to indulge in an abundance of food.

Among his contributions to the progress of medicine were special studies of the arthritic symptoms and the visceral neuroses in association with locomotor ataxia. He accordingly chose as the topic for the "Goulstonian Lecture," which he delivered in 1884 before the Royal College of Physicians, "The Neuroses of the Viscera," a subject that was only then beginning to be understood.

The Goulstonian Lectureship was founded in 1632 by Dr. Goulston, who bequeathed to the Royal College of Physicians £200 for the main-

16. Allbutt, Thomas Clifford: The Significance of Skin Affections in the Classification of Diseases, *St. George's Hosp. Rep.* 2:187-204, 1867.

17. Allbutt, Thomas Clifford: Histological Changes in Syphilitic Disease of the Cerebral Arteries, *St. George's Hosp. Rep.* 3:55-65, 1868.

tenance of an annual lectureship "to be read by one of the four youngest doctors in physic of the College, between Michaelmas and Easter in three days together both forenoon and afternoon for an honorarium of £10." Thus, for more than two hundred and fifty years the Goulstonian Lectureship had been handed out as a form of compliment to the youngest of the fellows reckoned on the basis of their election. Allbutt was then 58 years of age!

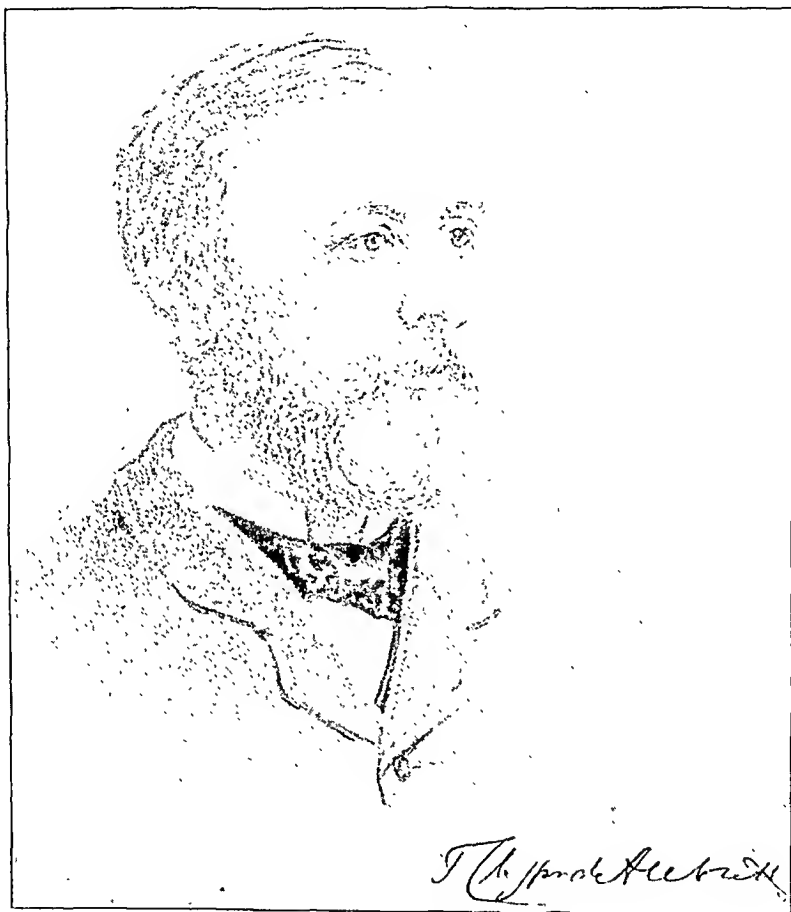


Fig. 11.—Allbutt in about 1880.

Desiring to be relieved somewhat of the burden of his enormous and steadily increasing consultation practice, with a prospect of greater leisure in which he might devote himself to wider things of the medical life, in 1889 he accepted the post of Commissionership in Lunacy, an official government position. At that day the salary of the most highly remunerative public medical appointment in England was £1,500. His professional life in Leeds accordingly came to a close, and he removed to London, where he continued to reside until 1892, when he was elected Regius Professor of Physic at Cambridge, at half the amount of salary

of his commissionership. Allbutt then removed to Cambridge, where he continued to reside until his death. From the day of his election, though over 50 years of age and without experience as a professor, he took his place in the front of university life. This ancient professorship, founded in 1540, had been held by a distinguished line of physicians including, among other notables, Francis Glisson, the anatomist, physiologist and pathologist.

Allbutt, who had been a member of the Royal College of Physicians since 1878, was elected a fellow in 1883, at the same time as William Osler, who was then only 33 years of age! The Royal College of Physicians, perhaps the most ancient society of physicians in Europe, received its charter from Henry VIII in 1578, having been founded by the King's physicians, of whom Linacre was the leader. It was a singular coincidence that Allbutt and Osler should each have been destined to achieve the high honor of the Regius Professorship, the one in Cambridge, the other in Oxford.

During his long career in medicine, covering more than sixty-four years, Allbutt received practically every honor that may come to a British physician. In 1866 he became a fellow of the Royal Medical and Chirurgical Society of London and contributed papers to its transactions. Besides his membership in British and foreign societies, from which he received honorary degrees and numerous titles of distinction, in public life he was consulting physician to many institutions.

In 1880, for his original work in clinical medicine the Royal Society bestowed on him the honor of fellowship, and he later served as vice president.

On June 27, 1904, in Convocation, before a brilliant assemblage in the Sheldonian Theatre, Oxford, the degree of Doctor of Science was conferred on him, in company with Jonathan Hutchinson, Sir Patrick Manson, William Osler and others.

He was associated also with societies and clubs which gave him the right to affix to his name the insignia of honors which England bestowed on the greatest of her men. In 1907 he was created Knight Commander of the Bath, and in 1920, Privy Councillor, the only man to be called to the Council as a physician, and was frequently called into consultation notably as a member of government commissions, especially that of the Home Office Inquiry into Trade Diseases. He was also Justice of the Peace in two counties. During the World War he served on several important military commissions and was honorary Lieutenant-Colonel of the Royal Army Medical Corps.

In 1896, he began to issue his great "System of Medicine," which was completed in eight volumes in 1899. Many of the articles in this edition were contributed by Allbutt himself. In 1896 there also appeared his "System of Gynecology," embodying the most recent advances in

the field culled from the literature of the world. In 1915 he finished "Diseases of the Arteries and Angina Pectoris." In this great work he expressed the theory that angina pectoris is due to disease of the root of the aorta. At first the idea was not received with much favor, yet in time it steadily gained adherence and is now believed by all.

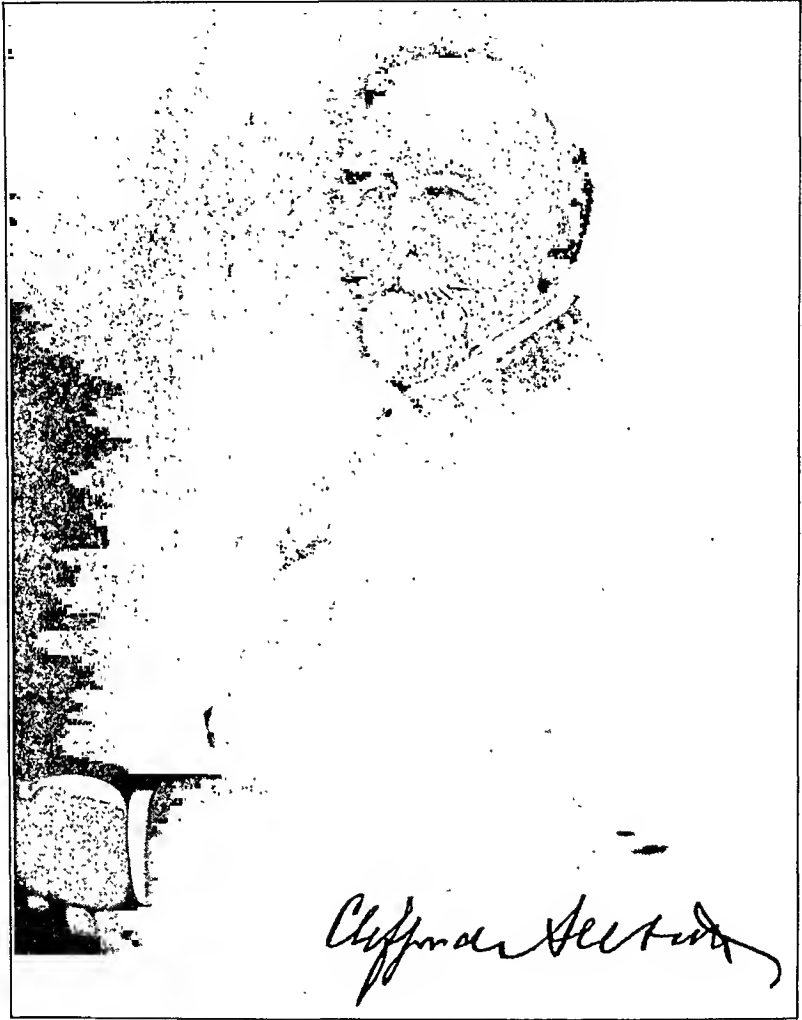


Fig. 12.—Allbutt in about 1899.

Another subject near to his heart which had for its object the consideration of the diseases of human beings and peoples and disease among animals was that of "Comparative Pathology," a subject which up to that time had been neglected, if not ignored. Allbutt urged that the manifestations of diseases and their effects on different individuals and races should be studied by comparison. He was so earnest an advocate of this extension of medical study that a section on this subject was formed in the Royal Society of Medicine, and Allbutt, appropri-

ately, was made the first president. Later he succeeded in establishing at Cambridge the now well known Institute of Comparative Pathology.

To the present generation Allbutt is best known, perhaps, as a medical writer with a pleasing yet powerful literary style. He was always more concerned with principles than with details. His scientific interests were varied; his contributions to literature all manifested the highest degree of scholarship. His knowledge of the literary side of medicine, both ancient and modern, was unrivaled and inexhaustible. Thinking and working along classical lines, like the true classical scholar that he was, he aptly and freely sprinkled quotations from Greek and Latin authors through the pages of his publications and in his correspondence.

His scholarship and wide knowledge of medical history served well to equip him to lecture on that subject. As a contributor to the literature on medical history he wrote papers and books on medieval science,¹⁸ surgery,¹⁹ Greek medicine in Rome²⁰ and Byzantine medicine.²¹ In 1902-1910 he delivered a number of addresses under the title of the Fitzpatrick Lectures on the history of medicine, at the Royal College of Physicians. A reading of his contributions on the "History of Medicine" in the eleventh edition of the "Encyclopaedia Britannica" will serve to show the wide scope of his knowledge.

He strongly held that good diction and literary style are as desirable in science as in literature and insisted that the character of one's writing "should be as carefully attended to as he felt that he himself was bound to express." As Regius Professor he had to read a great number of theses; in many of these, while he found the matter good he was vexed by defects of writing, which he described as "not mere inelegance, but such as to perplex and even travesty or hide the author's meaning." He therefore prepared a set of "Notes on the Composition of Scientific Papers," a work giving evidence of Allbutt's zeal in the education of physicians, for in it are carefully exposed the faults that writers are prone to commit. The book can serve as the memorial of a master of the most elegant English who descended to the task of teaching ordinary students how to write.

In September 1904, while on a visit to this country, he attended the meeting of the Congress of Fine Arts and Sciences, at the St. Louis

18. Allbutt, Thomas Clifford: *Science and Medieval Thought*, London, C. J. Clay & Sons, 1901.

19. Allbutt, Thomas Clifford: *Historical Relations of Medicine and Surgery to the End of the Sixteenth Century*, New York, Macmillan & Co., 1905.

20. Allbutt, Thomas Clifford: *Greek Medicine in Rome*, New York, The Macmillan Company, 1921.

21. Allbutt, Thomas Clifford: *Byzantine Medicine*, New York, The Macmillan Company, 1921.

has the good fortune to possess a person who, through extensive knowledge and acute judgment and by a phenomenal gift of expression is able to link the past with the present, and to record the history of medical science thereby becoming an important agent whose efforts make the objective sciences more humane." Such a man was Sir Thomas Clifford Allbutt.

Like Jonathan Hutchinson, Allbutt was a "forward-looking" man. In his Harveian Oration, "On Science and Medical Thought," delivered in October 1900, Allbutt declared: "We celebrate the memory of great men in the certain hope that in their children they will be born again." And at another time he stated: "We are living thankfully in a glorious time; our children will go much further still but they will owe much of their progress to the principles laid down by the great men of our own times."

Since Allbutt's bent was artistic rather than scientific, it was not expected by those who observed him in his early years that he would acquire a commanding reputation among physicians of the world. A quotation from an article in the *British Medical Journal* for 1883 epitomizes his creed: "The best doctor is the best artist and the best medical artist is the master and not the servant of his science."²³

The artistic element in his nature was never lost or even subdued but sought expression by his writings in which his artistic feeling played a part in his literary ability so manifest in his medical, scientific and historical writings, as well as in his occasional speeches in his later years.

Allbutt was a leader of the medical profession in England throughout his days; his position was unique in the medical world. A great physician, great scholar, great teacher and great writer, in his own generation he can be compared only with Sir William Osler, but in his finished literary style he surpassed the latter, as he did every one else. Sir Berkeley Moynihan paid this tribute to him at the University of Leeds (1923-1924):

Sir Clifford Allbutt, the most deeply learned physician of this day, master of a style of English which for sheer beauty and majesty is perhaps unmatched by that of any other scientific author of our generation; an Orator whose speech makes time seem hasty; a cultured upright English gentleman is the pride of the school he served so long and so well.

Allbutt readily appreciated the work of others and was always sympathetic toward all honest work, even though he might not feel free to commend the result. He was a keen critic of any doctrine brought forward, and his criticisms when called for were boldly stated and unflinching expressed. He possessed a remarkably flexible mind and was always progressive in his views and writings. To the last he kept

23. Allbutt, Thomas Clifford: *Brit. M. J.* 2:664, 1883.

abreast of the times and was ever ready to encourage other workers, pointing out to them where the labor might bear fruit. Up to the week before his death his mind was keenly alert on the salient points in any discussion in the contemporary medical journals, and he died in the full tide of his activities. He was to have lectured at the Royal Society in the autumn of 1925.

His frequent letters to the London *Times* served to endear him to the hearts of laymen, who found helpful advice and criticism in his contributions. In the number of the *Lancet* containing his obituary is a letter from him on "Alkalies in Certain Diseases of the Skin," evoked by a recent article in that journal. To the end he declared that "only two things are essential--to live uprightly and to be wisely industrious." The guiding star of his life, he used to say, was industry, and he declared that any little success that he might have had was due to constant application; yet, he thoroughly believed that a physician should take a continuous holiday of six weeks; he once missed doing so and regretted it ever after. He had traveled extensively, visiting Greece and Rome and other Italian cities along the Mediterranean, as well as Bayreuth and the Scandinavian countries.

For many years--indeed, from his fourteenth year and until he was in his ninetieth year--he had been famous as an alpinist and was well known in the north of England, especially in the lake region, for his long walks through that rough country. Osler, in 1918, recorded that he found Sir Clifford in fine form and that despite his 82 years he was exhibiting extraordinary physical power, thinking nothing of cycling 15 miles (24.2 kilometers) daily! Except for deafness, age seemed not to affect his faculties, while his subtle intellect was keen to the end of his long life.

Clifford Allbutt was distinguished in looks as well as in activities. He had a wonderful ability to deal with men and things, and every one who came in contact with him was deeply impressed by his keen intelligence, culture and urbanity. He was often referred to as the "spiritual aristocrat of medicine." His courtesy was unfailing to all, high or low, and, like other qualities, was a finished product. In his personal relations he was one of the kindest of men, and despite the exalted position to which he was placed by acclamation of his fellows he was to his last hours without a trace of that arrogance which success engenders in lesser minds. In spite of all this he must have realized that he had become one of the most honored and beloved of the heads of the medical profession and had largely contributed to the advancement of medical science. Like the true Yorkshireman that he was, he was conscious of his own worth, yet so gracious and urbane was he that one would never have suspected him of being one of that "peculiar" county, although under the polish the grit was there.

He was a much desired conversationalist and lecturer. His home was the center of scientific and literary exchange, as he was well acquainted with many famous authors, both general and scientific. He was on intimate friendly terms with the Brontë sisters, and he was supposed to have inspired the character Tertius Lydgate in George Eliot's "Middlemarch." Osler,²⁴ in commenting on this, declared that although George Eliot had frequent occasions for contact with Sir Clifford, there was nothing in his career in common with that of the famous character except their training and their high aspirations. It is likely that Allbutt's early course at Leeds had given the gifted authoress a suggestion for the portrayal of a character in her novel.

When one contemplates the activities of this long life one is struck by Allbutt's deep and all-absorbing interest in ophthalmology and his giving out to the world for many years the product of his study. It was an episode in his life—a mere incidental reaction to the call for an understanding of great medical problems. That he did not continue with the special practice of ophthalmology when the time in his affairs seemed ripe for him to confine himself to that branch is an unsolved mystery. That he should have accomplished what he did exemplifies the well known characteristics of Yorkshire perseverance and strenuous devotion to whatever work one has at hand. Allbutt was quick to seize the opportunity offered by Dr. Ogle's suggestion that the ophthalmoscope might be of service in the diagnosis of intracranial disorders. Allbutt was never afraid of work, and despite the unwillingness of many practitioners to assist and their timidity to persist in studying the eyes he was never doubtful of success and never weary in carrying out whatever task he undertook.

In his interest in ophthalmology Allbutt might be likened to one of the early navigators who ventured out in voyaging to an unexplored land. There was a "point of departure," but no "course and distance" could be defined prior to his sailing. A "landing" was to be made somewhere, yet because his instruments were imperfect, much of navigation was accomplished by "plane sailing." But, like the captain, Allbutt, being a careful navigator, made frequent "observations" to ascertain his position; the results he "plotted" from time to time before the voyage was over; a "track" was lined off on the "chart" and the details recorded in his "log book." A true course having been laid, it was possible for him, as for the captain, to return to the point of departure, and a good account was given of the voyage, with a descrip-

24. Osler, cited in Cushing, Harvey: *Life of Sir William Osler*, Oxford, Clarendon Press, 1925, vol. 1, p. 463.

tion of the newly explored country. Allbutt's ophthalmoscope was his "observation-instrument"; his discussions and writings became his chart and his log book.

So satisfied was this "navigator" with the product of his voyage and so willing was he that others should share in his rewards that he widely opened the way for any who wished to make the same explorations. Although he himself never set out again for his early field, what he learned by his sailing he faithfully applied in all his later voyaging.

Present day ophthalmologists, Allbutt's successors and followers, are the later voyagers into that continent so freely opened out to them. His unwillingness to pursue ophthalmology as a source of special medical practice is one of the remarkable phenomena in the history of modern medicine. I have undertaken this memoir lest the part he played in this drama might become forgotten by ophthalmologists in this generation and the next.

The materials for this memoir were gathered from many sources and accumulated over a number of years. In the removal of my library from one house to another, in March, the list of references was mislaid and has not been recovered. Each of Allbutt's published papers and reports and the comments and discussions offered by contemporaries were read and analyzed; the epitome was included in what appears here. I failed to find Allbutt's name in the index of Hirschberg's "*Geschichte der Augenheilkunde*"²⁵ and in Wilbrand and Saenger's "*Die Neurologie des Auges*."²⁶

I had never met Sir Clifford and had only a brief correspondence with him. I therefore have had to rely on numerous personal letters received from Sir James Crichton-Browne and others who had known him. Much was taken from Sir Humphry Davy Rolleston's "Memoir."

The object of this series of "Short Studies on the History of Ophthalmology" has been to tell of the introduction of ophthalmoscopy into medical practice and to present memorials of those who did so much through ophthalmoscopic investigations in the creation of modern medicine. So far as concerned England, the earliest days of the ophthalmoscope have been recounted and the lives of Hutchinson, Hughlings Jackson and Clifford Allbutt presented. The status of their contribution

25. Hirschberg, J.: *Geschichte der Augenheilkunde*, in von Graefe, T., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, Leipzig, W. Engelmann, 1915.

26. Wilbrand, H., and Saenger, A.: *Die Neurologie des Auges*, Wiesbaden, J. F. Bergmann, 1900.

might be expressed by the following testimony from Dr. W. H. Broadbent,²⁷ Physician to St. Mary's Hospital, London:

The ophthalmoscope, thanks to the efforts of Dr. Clifford Allbutt and Dr. Hughlings Jackson, may be said fairly to have taken its place in English medicine; and it is scarcely possible to over state the additional interest it has brought into the study of the diseases of the nervous system. It has removed causes of confusion and error; it has opened absolutely new sources of information as to the state of the intracranial circulation, which will eventually conduce greatly to decision in treatment. It has, also, or rather the far-reaching vision of the men who have forced it upon the attention of the profession, widened the horizon of pathological research by stating new questions of various kinds for solution.

317 South Fifteenth Street.

27. Broadbent, W. H.: On the Causation and Significance of the Choked Disk in Intracranial Diseases, *Brit. M. J.* 1:633, 1872.

EFFECT OF FATIGUE ON THE ADJUSTMENT OF THE EYE TO NEAR AND FAR VISION

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U. S. S. PORTLAND

NAV. MED. CAMP

In previous work done with the tachistoscope in the measurement of the speed of adjustment of the eye to near and far vision I¹ reported only the normal findings. Since that time I have been able to accumulate data for tests of the dynamic speed of vision for pilots with fatigue after one, two, three and four hours of flying.²

I shall first show the results of six hundred and twenty-eight tests on aviators under normal conditions in the three phases of speed of accommodation, i. e., the speed of accommodation from near to far, the speed of accommodation from far to near, and the speed of accommodation from near to far and return to near.

This is to demonstrate what I consider the normal limits for subjects arranged according to age in five groups and to show the causes for the data which are not within those limits.

In all the tables in this paper the pilots are arranged in five year groups progressing from 20 to 50 years of age, as noted in the left column of the tables. The time is given in fractions of seconds, in progressions of one-tenth second from left to right at the top of tables 1 to 8. The average time for each five year group and the number of tests for each five year group are noted in the last two right columns of tables 1 to 8. The total number of tests carried out in each group of 0.1 second is noted at the bottom of the tables.

The total number of tests and the average time for all the age groups are noted in the right lower corner of tables 1 to 8.

* In care of Postmaster.

1. Robertson, C. J.: (a) Measurement of the Speed of Adjustment of the Eye to Near and Far Vision, U. S. Nav. M. Bull. **32**:275-283 (July) 1934; (b) A Comparative Study of the Measurement of the Speed of Adjustment of the Eye for Near and Far Vision, *ibid.* **33**:187-205 (April) 1935; (c) Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82-89 (July) 1935; (d) Measurement of Speed of Adjustment of Eye to Near and Far Vision: A Further Study, *ibid.* **15**:423-434 (March) 1936.

2. (a) Ferree, C. E., and Rand, G.: An Instrument for Measuring Dynamic Speed of Vision, Speed of Accommodation, and Ocular Fatigue, Arch. Ophth. **15**: 1072-1687 (June) 1936. (b) Aviation Physical Examination, United States Navy Standard, 1927, in the Manual of the Medical Department, United States Navy, Washington, D. C., Government Printing Office, 1927.

six cases. The pathologic conditions were: inequality of visual acuity, inequality of accommodation, astigmatic error and exophoria of 10 diopters at 33 cm.

In the group of subjects from 25 to 29 years old, inclusive, no test carried out in the time range beyond 0.7 second showed pathologic conditions.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination in the time range beyond 0.8 second showed exophoria of 10 diopters at 33 cm. in two cases.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination in the time range beyond 0.9 second showed visual acuity of less than 20/20 in two cases.

TABLE 3.—*Speed of Accommodation of Pilots from Near to Far and Return to Near*

Age, Years	Time, Seconds										Num- ber of Tests	Average Time for Group, Seconds
	0.95 to 1.049	1.05 to 1.149	1.15 to 1.249	1.25 to 1.349	1.35 to 1.449	1.45 to 1.549	1.55 to 1.649	1.65 to 1.749	1.75 to 1.849	1.85 On		
20 to 24	7	17	33	24	9	90	1.171
25 to 29	21	40	102	95	36	9	2	305	1.239
30 to 34	1	9	27	48	35	26	5	4	1	1	157	1.329
35 to 39	..	2	7	11	12	9	8	6	1	1	57	1.421
40 to 44	1	1	2	2	4	1	3	14	1.663
45 to 49	1	1	3	5	2.323
Total	29	68	169	179	93	46	17	15	4	8	628	
Average time for all age groups, seconds.....												1.526

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination in the time range beyond 1 second showed pathologic conditions in one case. These were exophoria of 10 diopters at 33 cm. and inequality of accommodation.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination in the time range beyond 1.1 seconds showed pathologic conditions in two cases. The conditions were: astigmatic error in one eye, astigmatism in each eye and exophoria of 10 diopters at 33 cm.

Table 3 shows the speed of accommodation from near to far and return to near. The maximum number of tests is found to be in the time range of from 1.25 to 1.349 seconds. The progressive increase of time, as well as the average time, is demonstrated for each five year group. The average time for all the age groups is 1.526 seconds.

The tests listed at the right of the heavy line are forty-six in number and are considered to have shown pathologic conditions. The

number of these cases in each age group and the types of conditions are taken up. Each subject may have one or more of the conditions noted.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from near to far and return to near in the time range beyond 1.35 seconds showed pathologic conditions in nine cases. The conditions were: exophoria of from 8 to 12 diopters at 33 cm., left hyperphoria near the extreme limit, inequality of visual acuity, unequal accommodation, astigmatism, visual acuity of less than 20/20 and myopia.

In the group of subjects from 25 to 29 years old, inclusive, the tests for the determination in the time range beyond 1.45 seconds showed pathologic conditions in eleven cases. The conditions were: exophoria of 10 diopters or more at 33 cm., astigmatic error, inequality of accommodation, myopia, visual acuity of less than 20/20 and inequality of visual acuity.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination in the time range beyond 1.55 seconds showed pathologic conditions in eleven cases. The conditions were: exophoria of from 8 to 10 diopters at a distance of 33 cm., exophoria of 10 diopters or more at 33 cm., astigmatism, inequality of accommodation and visual acuity of less than 20/20.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination in the time range beyond 1.65 seconds showed pathologic conditions in eight cases. The conditions were: inequality of visual acuity, astigmatism and visual acuity of less than 20/20 in one or both eyes.

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination in the time range beyond 1.75 seconds showed pathologic conditions in four cases. The conditions were: exophoria of 10 diopters at 33 cm., astigmatic error and exophoria of 12 diopters at 33 cm.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination in the time range beyond 1.85 seconds showed pathologic conditions in three cases. The conditions were: visual acuity of less than 20/20 cm. and exophoria of 14 diopters at 33 cm.

In a brief résumé of the cases in which the speed of accommodation to near and far vision was considered below the limits of safety the cause for the loss in speed is deemed evident and proved. It is considered that as age progresses the pilots with known ocular conditions are less and less able to overcome the conditions and thus become an aeronautic menace. Certain conditions can be improved with proper treatment.

It is noted that many pilots have a pathologic condition of the eyes but do not show a loss in speed of accommodation. These pilots are in the younger age groups, and it is considered that by virtue of their youth they are able to overcome the condition, but they should be carefully watched as age progresses.

Of the visual requirements in aviation as laid down in the Manual of the Medical Department of the United States Navy,^{2b} the following conditions did not show in my tests as pathologic: esophoria and exophoria at a distance of 6 meters, prism divergence and depth perception.

It is considered that the following conditions definitely cause a slowing of the speed of adjustment of the eye to near and far vision: age; exophoria of 8 diopters or more at 33 cm.; astigmatism of one or both eyes; visual acuity below 20/20 in one or both eyes; inequality of visual acuity, even though vision is above the required 20/20; myopia, an angle of convergence near the lower limits of normal and a sluggish mental reaction.

In table 4 is shown the speed of accommodation from near to far and return to near of pilots after one hour of flying compared with their normal speed of accommodation. It is not considered that one hour of flying causes severe fatigue; nevertheless it is demonstrated that even with the minor degree of fatigue resulting, there is a slowing of the speed of adjustment of the eye to near and far vision. The effect of fatigue is demonstrated throughout the table, with particular reference to the average time for the age groups.

The cases in which the speed of accommodation is considered below the limit of safety are discussed separately, and the suspected cause for the slowing is given.

In the group of subjects from 20 to 24 years old, inclusive, tests for determining the speed of accommodation from near to far and return to near showed a speed below the limits of safety in four cases.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.352 seconds, and speed in the fatigued state, 1.452 seconds.

CASE 2.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.353 seconds.

CASE 3.—Astigmatic error, visual acuity of less than 20/20, left hyperphoria of 0.75 diopter, and exophoria of 12 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.386 seconds, and speed in the fatigued state, 1.562 seconds.

CASE 4.—Astigmatic error and inequality of visual acuity were demonstrated. The tachistoscopic findings were: normal speed, 1.32 seconds, and speed in the fatigued state, 1.352 seconds.

In the group of subjects from 25 to 29 years old, inclusive, tests for the determination showed speed below the limits of safety in five cases.

TABLE 4.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After One Hour of Flying Compared with Their Normal Speed*

Age, Years	Time, Seconds																Num- ber of Tests	Average Time for Age Group, Seconds					
	0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549		1.55 to 1.649		1.65 to 1.749				1.75 On				
	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State	Nor- mal State	Fa- tigue d mal State			Nor- mal State	Fa- tigue d mal State			
20 to 24	1	1	6	5	9	9	12	12	3	2	..	1	..	1	31	1,231	1,234		
25 to 29	3	1	11	7	34	21	23	32	7	15	3	5	81	1,214	1,237		
30 to 34	..	1	2	1	9	2	6	12	11	10	..	1	2	50	1,296	1,262		
35 to 39	1	1	3	1	2	1	..	3	3	2	3	2	1	..	10	1,412	1,507		
40 to 44	1	1	..	1	1,225	1,623		
45 to 49	1	1	1	1,782	2,461		
Normal	4	19	53	41	33	57	23	23	3	3	1	13	3	1	2	2	151	1,492	1,575				
Fatigued	3	13	13	57	33	57	23	23	3	3	1	13	1	1	2	2	151	1,492	1,575				
Average time for all age groups, seconds.....																						1,492	1,575

TABLE 5.—*Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Two Hours of Flying Compared with Their Normal Speed*

[illegible]

CASE 1.—Visual acuity was 20/20 with effort. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.485 seconds.

CASE 2.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.21 seconds, and speed in the fatigued state, 1.485 seconds.

CASE 3.—Exophoria of 10 diopters at 33 cm. was present. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.507 seconds.

CASE 4.—Inequality of accommodation and exophoria near the outside limits at a distance of 33 cm. were demonstrable. The tachistoscopic findings were: normal speed, 1.453 seconds, and speed in the fatigued state, 1.496 seconds.

CASE 5.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.468 seconds, and speed in the fatigued state, 1.452 seconds.

In the group of subjects from 35 to 39 years old, inclusive, tests for the determination showed speed below the limits of safety in two cases.

CASE 1.—Right hyperphoria of 0.75 diopters was present. The tachistoscopic findings were: normal speed, 1.672 seconds, and speed in the fatigued state, 1.76 seconds.

CASE 2.—There was no record of findings due to age. The tachistoscopic findings were: normal speed, 1.672 seconds, and speed in the fatigued state, 1.804 seconds.

In the group of subjects from 45 to 49 years old, inclusive, the tests for the determination showed speed below the limits of safety in 1 case.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.782 seconds, and speed in the fatigued state, 2.464 seconds.

Chart 1 shows the speed of accommodation from near to far and return to near of pilots with fatigue after one hour of flying compared with their normal speed of accommodation in terms of the average time for the five year age groups. Only one test was carried out in the group of subjects from 40 to 44 years old, inclusive, and only one in that of subjects from 45 to 49 years old, inclusive. Even with this minor degree of fatigue (after one hour of flying) the slowing of speed is well noted.

Table 5 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying compared with their normal speed of accommodation. It is here more evident that fatigue has a bearing on the speed of adjustment for near and far vision.

In the group of subjects from 20 to 24 years old, inclusive, the tests for determining the speed of accommodation from near to far and return to near showed a speed below the limits of safety in one case.

CASE 1.—Inequality of visual acuity and hyperopia were present. The tachistoscopic findings were: normal speed, 1.309 seconds, and speed in the fatigued state, 1.441 seconds.

In the group of subjects from 25 to 29 years old, inclusive, the tests for the determination showed a speed below the limits of safety in six cases.

CASE 1.—Myopia was present. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.54 seconds.

CASE 2.—Inequality of accommodation and exophoria of 10 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.309 seconds, and speed in the fatigued state, 1.45 seconds.

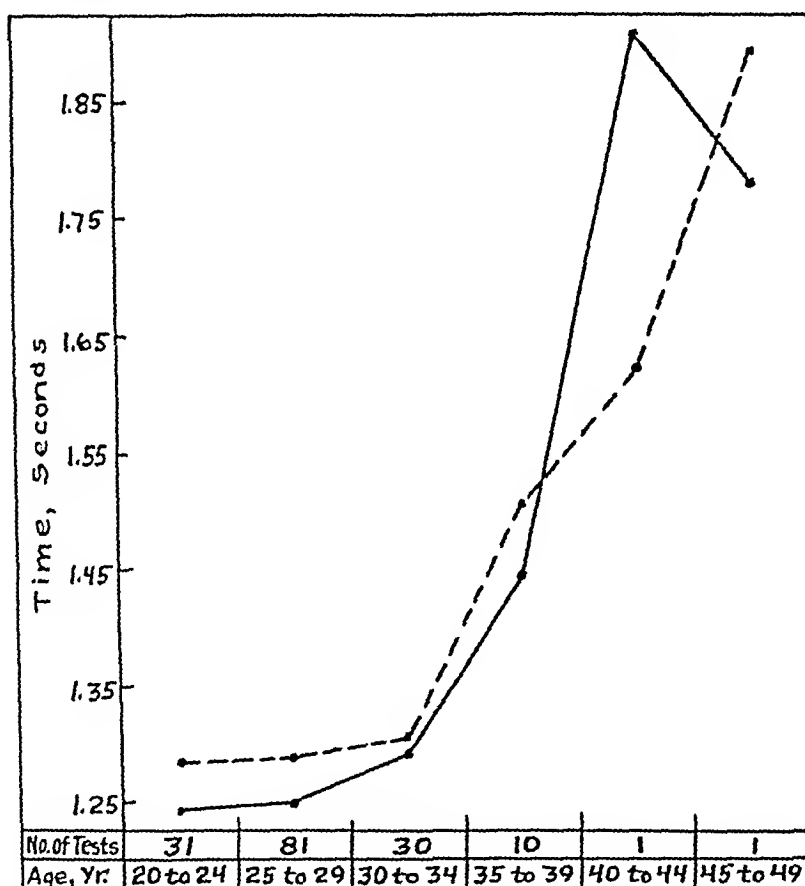


Chart 1.—Speed of accommodation from near to far and return to near of pilots with fatigue after one hour of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

CASE 3.—Exophoria of 10 diopters at 33 cm. was present. The tachistoscopic findings were: normal speed, 1.287 seconds, and speed in the fatigued state, 1.507 seconds.

CASE 4.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.353 seconds, and speed in the fatigued state, 1.551 seconds.

CASE 5.—Myopia was found. The tachistoscopic findings were: normal speed, 1.408 seconds, and speed in the fatigued state, 1.606 seconds.

CASE 6.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.496 seconds, and speed in the fatigued state, 1.496 seconds.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination showed a speed below the limits of safety in three cases.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1,364 seconds, and speed in the fatigued state, 2,277 seconds.

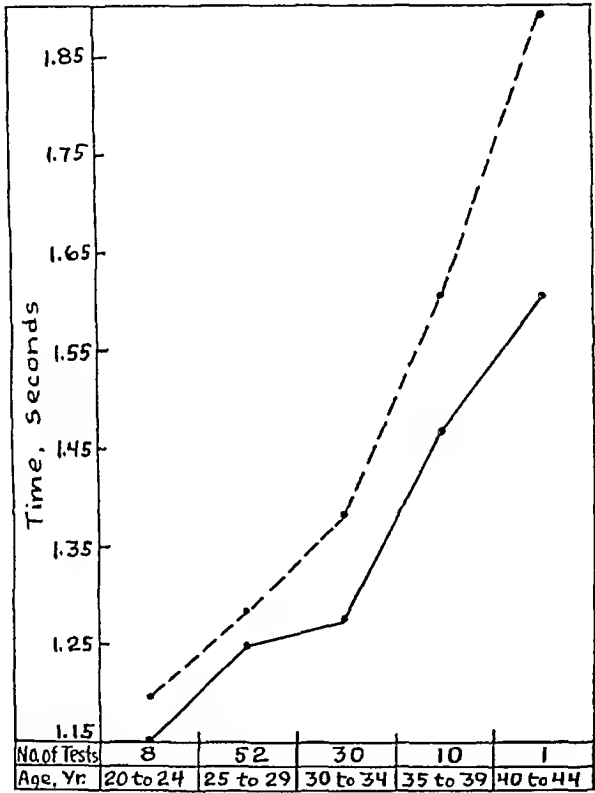


Chart 2.—Speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

CASE 2.—No pathologic condition was demonstrable. The tachistoscopic finding were: normal speed, 1.716 seconds, and speed in the fatigued state, 1.87 seconds.

CASE 3.—Inequality of visual acuity and exophoria of 9 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.606 seconds.

In the group of subjects from 35 to 39 years old, inclusive, the test for the determination showed a speed below the limits of safety in one case.

TABLE 6.—Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Three Hours of Flying Compared with Their Normal Speed

Age, Years	Time, Seconds												Num- ber of Tests	Average Time for Age Group, Seconds							
	0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549			1.55 to 1.649		1.65 to 1.749		1.75 On			
	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State		Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State		
20 to 24		
25 to 29	3	1	..	1	1	2	4	1.121 1.231		
30 to 34	1	1	4	1	6	6	1	2	2	2	1	3	15	1.321 1.330		
35 to 39	1	1	1	..	1	1	1	1	..	1	1	5	1.470 1.575		
40 to 44		
45 to 49		
Normal Fatigued	4	2	5	3	8	3	2	3	3	3	1	4	..	1	1	24	1.304 1.396		
Average time for all age groups, seconds,.....																				1.304	1.396

TABLE 7.—Speed of Accommodation from Near to Far and Return to Near of Pilots with Fatigue After Four Hours of Flying Compared with Their Normal Speed

Age, Years	Time, Seconds												Num- ber of Tests	Average Time for Age Group, Seconds						
	0.95 to 1.049		1.05 to 1.149		1.15 to 1.249		1.25 to 1.349		1.35 to 1.449		1.45 to 1.549			1.55 to 1.649		1.65 to 1.749		1.75 On		
	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State		Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	Nor- mal State	Fa- tigue mal State	
20 to 24	
25 to 29	2	2	1	1	2	1	1.234	1.242	
30 to 34	..	1	..	2	1	1	1	3	1	1	1	1	1.286	1.364	
35 to 39	1	1	1	1	1.424	1.485	
40 to 44	1	1.542	1.683	
45 to 49	
Normal	..	1	2	4	2	4	4	4	1	2	2	1	2	1	1	
Fatigued	13	
Average time for all age groups, seconds.....																			1.370	1.442

Inequality of visual acuity and vision of less than 20/20 were found. The tachistoscopic findings were: normal speed, 1.595 seconds, and speed in the fatigued state, 1.716 seconds.

In the group of subjects from 40 to 44 years old, inclusive, the tests for the determination showed a speed below the limits of safety in one case.

Inequality of accommodation and exophoria of 9 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 1.595 seconds, and speed in the fatigued state, 1.892 seconds.

Chart 2 shows the speed of accommodation from near to far and return to near of pilots with fatigue after two hours of flying compared with their normal speed of accommodation, in terms of the average time

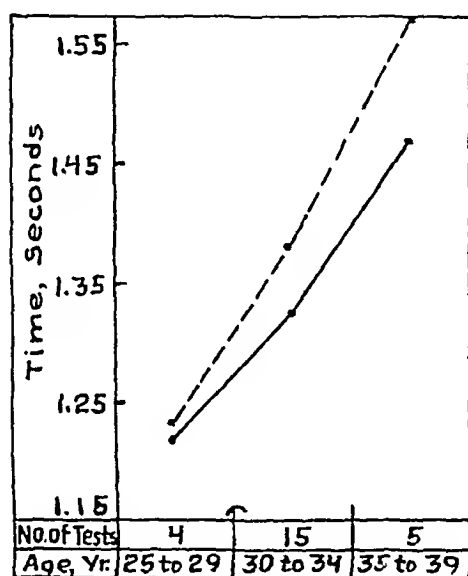


Chart 3.—Speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

for each five year age group. This demonstrates the more noted slowing of the speed with greater fatigue, particularly in the later age groups.

Table 6 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying compared with their normal speed of accommodation. Again the effect of fatigue is shown.

In the group of subjects from 30 to 34 years old, inclusive, the tests for the determination showed a speed below the limits of safety in three cases.

CASE 1.—No pathologic condition was demonstrable. The tachistoscopic findings were: normal speed, 1.496 seconds, and speed in the fatigued state, 1.617 seconds.

CASE 2.—Astigmatic error, inequality of visual acuity and vision of less than 20/20 in one eye were present. The tachistoscopic findings were: normal speed, 1.573 seconds, and speed in the fatigued state, 1.551 seconds.

CASE 3.—Inequality of accommodation and exophoria of 9 diopters at 33 cm. were found. The tachistoscopic findings were: normal speed, 1.43 seconds, and speed in the fatigued state, 1.617 seconds.

In the group of subjects from 35 to 39 years old, inclusive, the tests for the determination showed a speed below the limits of safety in one case.

CASE 1.—Visual acuity of less than 20/20, inequality of visual acuity and exophoria of 10 diopters at 33 cm. were present. The tachistoscopic findings were: normal speed, 2.2 seconds, and speed in the fatigued state, 2.002 seconds.

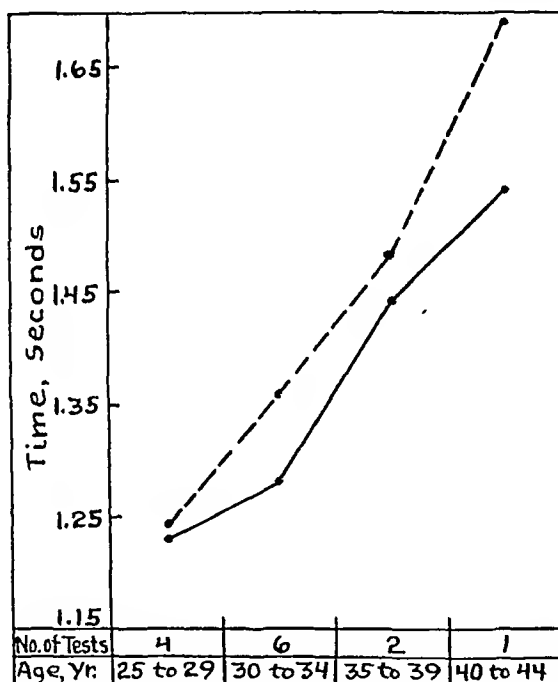


Chart 4.—Speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

Chart 3 demonstrates the speed of accommodation from near to far and return to near of pilots with fatigue after three hours of flying with their normal speed, in terms of the average time for the five year age groups.

Table 7 shows the speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying compared with their normal speed of accommodation. As there were only thirteen tests in this study it is difficult to draw conclusions of moment. However, the effect of fatigue is definitely demonstrated.

Chart 4 shows the speed of accommodation from near to far and return to near of pilots with fatigue after four hours of flying compared with the normal speed of accommodation of the same pilots, in terms of the average time for the five year age groups.

Chart 5 shows the speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying compared with their normal speed of accommodation, in terms of the average time for the five year age groups.

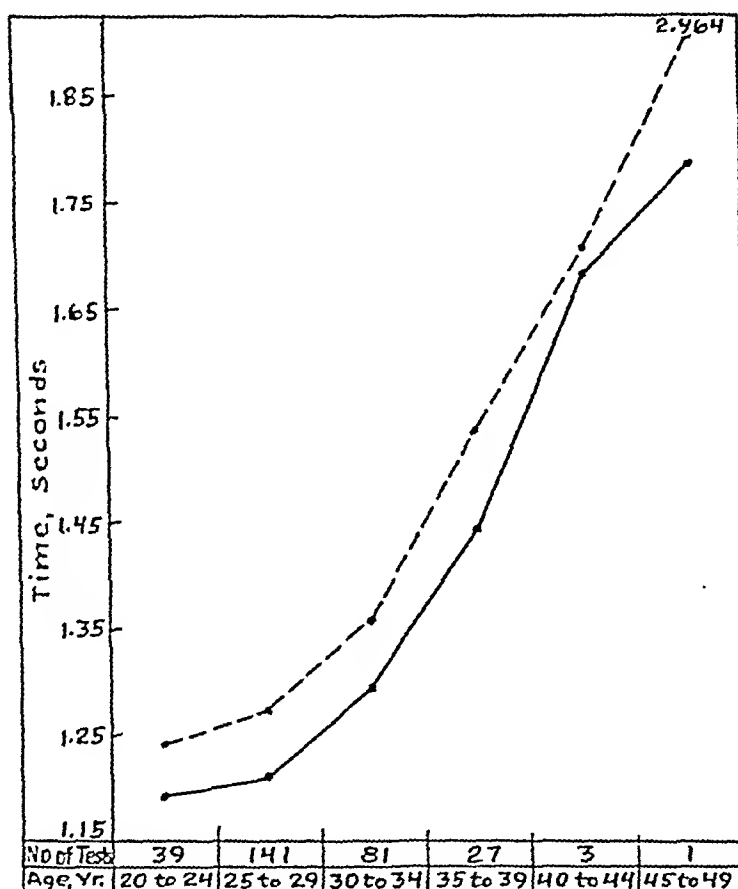


Chart 5.—Speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying (broken line) compared with their normal speed of accommodation (unbroken line), in terms of the average time for the five year age groups.

Tables 9, 10 and 11 show the average speed of accommodation from near to far, from far to near and from near to far and return to near, respectively, in seconds or fractions of seconds of pilots under normal conditions and pilots with fatigue after one, two, three and four hours of flying compared with their normal speed of accommodation in terms of the average time for the 5 year age groups. There is noted at the bottom of each table the average time for all the age groups under each type group.

TABLE 10.—*Speed of Accommodation from Far to Near of Pilots and Nonpilots and of Pilots with Fatigue After One, Two, Three and Four Hours of Flying and Under Normal Conditions, in Terms of the Average for Each Age Group*

[illegible]

TABLE 11.—Speed of Accommodation from Near to Far and Return to Near of Pilots and Nonpilots and of Pilots with Fatigue After One, Two, Three and Four Hours of Flying and Under Normal Conditions, in Terms of the Average for Each Age Group

Age, Years	Speed of Pilots, Sec.	Num- ber of Tests	Speed of Non- pilots, Sec.	Num- ber of Tests	Speed After 1 Hr. of Flying, Sec.	Num- ber of Tests	Nor- mal Speed	Speed After 2 Hrs. of Flying, Sec.	Num- ber of Tests	Nor- mal Speed	Speed After 3 Hrs. of Flying, Sec.	Num- ber of Tests	Nor- mal Speed	Speed After 4 Hrs. of Flying, Sec.	Num- ber of Tests	Nor- mal Speed	Speed After 1 to 4 Hrs. of Flying, Sec.	Num- ber of Tests	Nor- mal Speed	Total Number of Tests for Age Group
20 to 24	1.171	90	1.235	53	1.284	31	1.231	1.197	8	1.150	1.240	39	1.190	221
25 to 29	1.239	305	1.327	21	1.287	81	1.244	1.287	52	1.251	1.231	4	1.121	1.242	4	1.234	1.274	141	1.212	608
30 to 34	1.329	157	1.380	22	1.303	30	1.296	1.339	30	1.271	1.350	15	1.321	1.361	6	1.283	1.345	81	1.293	341
35 to 39	1.421	57	1.625	5	1.507	10	1.443	1.535	10	1.444	1.575	5	1.470	1.485	2	1.424	1.538	27	1.445	116
40 to 44	1.608	14	1.676	10	1.628	1	1.925	1.892	1	1.595	1.683	1	1.590	1.701	3	1.686	30
45 to 49	2.232	5	1.782	1	2.464	1	1.782	2.464	1	1.782	8
Total.....		628		112		159			101			24			13			292		
Total aver.	1.691		1.509		1.578		1.492	1.459		1.341	1.396		1.430	1.492		1.370	1.456		1.408	1,324

The findings for one hundred and twelve nonpilots have been added in these tables merely for the sake of interest and comparison.

Table 8 shows the speed of accommodation from near to far and return to near of pilots with fatigue after from one to four hours of flying compared with their normal speed of accommodation. The effect of fatigue is readily noted in the time and age groups and in the average time of each five year age group.

No consideration is given here to the cases in which the speed was below the limit of safety, as they were incorporated under the separate demonstrations of the speed of accommodation of pilots with fatigue after one, two, three and four hours of flying.

The same precautions were taken in these tests as in previous tests, as noted in my other articles.¹ The illuminometer was used to check illumination and the tachometer to check the speed of the motor. All the tests were carried out by one technician, so that there was no variation in the technic.^{1b}

SUMMARY

In previous articles¹ and in the beginning of this article an attempt has been made to establish a standard of speed within safe limitations in five year age groups of the three phases of adjustment of the eye to near and far vision with particular reference for use in aviation. The reasons, including age, for the speed of accommodation's being outside these limitations have been demonstrated.

In the latter part of this article the effect of fatigue is demonstrated, and tables showing the speed of accommodation under normal conditions compared with the speed of accommodation in the fatigued state are given.

CONCLUSIONS

Age has a marked bearing on the speed of adjustment of the eye to clear seeing at different distances.

With any anomalous conditions of the eye the factor of age becomes of greater importance.

After the age of 30 the change becomes rapidly more marked.

Tables 1, 2 and 3 demonstrate what I consider the limits allowable in the three phases of the adjustment of the eye—that from near to far, that from far to near and that from near to far and return to near.

In tables 1, 2 and 3 are demonstrated the variations of the speed of adjustment.

Various anomalous conditions of the eye such as were noted in the discussion of the tests that demonstrated speeds below the limits of safety are, in part, the cause of the slowing of the speed.

Aviators with speed of adjustment not within the limits of safety should be disqualified physically until such time as adjustment can be made, or permanently if it is found that they cannot make the adjustment.

Fatigue has a marked bearing on the speed of adjustment in all ages, more notably so as age progresses.

Fatigue other than that caused by flying can be tested by this method.

The study of fatigue and of the time of recovery is an important phase of this work as yet untouched.

ANTERIOR CAPSULAR CATARACT

AN EXAMPLE OF TRUE METAPLASIA

HARVEY D. LAMB, M.D.

ST. LOUIS

The particular nature of the so-called anterior capsular cataract has never been exactly defined. Regarding its pathogenesis, there exists less difference of opinion. As the name indicates, proliferation of the cells belonging to the anterior epithelium of the lens has been generally accepted as the essential factor in the formation of the anterior capsular cataract. However, the nature of the tissue formed from this proliferation of the epithelial cells has been only suggested.

Beer¹ in 1817 described the clinical appearance of anterior capsular cataract. Müller² in 1857 discussed the histologic changes and concluded that the new-formed layers show transition to masses of anomalous structure, for example, fibrous tissue. The English pathologists Parsons³ and Collins and Mayou⁴ described the tissue forming the anterior capsular cataract as being composed of flattened epithelial cells and their processes. Hess⁵ mentioned only the proliferation of the epithelial cells in the histologic observations. Jess⁶ stated that the proliferated epithelial cells are separated from the capsule by many layers of a structure resembling connective tissue. Peters,⁷ from the study of a number of cases of anterior capsular cataract, concluded that there exists a true epithelial capsular cataract and that organized connective tissue occurs inside the capsular sac only when it proliferates through a rupture in the capsule of the lens.

Axenfeld,⁸ while discussing the possibility of the transition of proliferated pigmented retinal epithelium into connective tissue, wrote that

1. Beer, G. J.: *Lehre von den Augenkrankheiten*, Vienna, Heubner & Volke, 1817, vol. 2, p. 289.

2. Müller, H.: *Ueber die anatomischen Verhältnisse des Kapselstaars*, Arch. f. Ophth. **3**:55, 1857.

3. Parsons, J. H.: *The Pathology of the Eye*, New York, G. P. Putnam's Sons, 1905, vol. 2, p. 407.

4. Collins, E. T., and Mayou, M. S.: *Pathology and Bacteriology of the Eye*, Philadelphia, P. Blakiston's Son & Co., 1925, p. 596.

5. Hess, C., in von Graefe, T., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 3, Leipzig, Wilhelm Engelmann, 1911, pt. 2, chap. 6, p. 217.

6. Jess, A., in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 5, p. 228.

7. Peters, A.: *Zur Kenntnis des Kapselstaars*, Arch. f. Ophth. **105**:154, 1921.

8. Axenfeld, T.: *Retinitis externa exudativa mit Knochenbildung im sehfähigen Auge*, Arch. f. Ophth. **90**:452, 1915.

in the case of proliferations of the epithelium of the capsule of the lens and in the case of capsular cataract, fibrous masses arise which present the characteristics of connective tissue by Van Gieson's and other methods of staining.

In a large collection of human eyeballs anterior capsular cataract is sufficiently common to permit the study of all stages of its formation. From a histologic examination of more than fifty lenses with this condition, I am convinced that anterior capsular cataract is composed of collagenous connective tissue, the fibroblasts of which are directly derived from the epithelial cells of the lens.

The microscopic observations on two patients presenting this change from epithelial cells to fibrous tissue will be briefly described.

OBSERVATIONS IN TWO CASES

CASE 1.—A man 51 years old noted the loss of all vision in his eye following a spell of coughing. When first examined at the ophthalmic clinic of the Washington University School of Medicine about one month later, this eye presented diminished tension, tremor of the iris and lens, a small degree of clouding in the lens and a grayish reflex from the fundus. Secondary glaucoma developed, making enucleation necessary after nine months because of severe pain. Examination of sections through the enucleated eye confirmed the clinical diagnosis of retinal detachment and dislocation of the lens.

The lens was rounded and presented a diffuse irregularly arranged liquefaction of its cortex. In the middle of the anterior surface of the lens there was observed a small, thin anterior capsular cataract. The part of the capsule of the lens covering the latter was much wrinkled, and on its anterior surface lay a thin layer of posterior synechial tissue and pupillary membrane. From the margin of the pupil adherent to the lens pigmented epithelium from the iris had proliferated for a short distance onto the anterior surface of the pupillary membrane. Since this thin layer of connective tissue on the anterior capsule did not extend beyond the limits of the capsular cataract, it may be concluded that the latter was probably due to the membrane on the surface of the capsule of the lens.

Under the wrinkled lens capsule over the anterior capsular cataract a small amount of albuminous fluid lay in the angles formed by the wrinkles. An occasional thin disk-shaped cell was observed in this fluid. The capsular cataract proper was composed of many thin lamellae arranged with their flat surfaces in a frontal plane or parallel to the anterior surface of the lens. Between these lamellae lay numerous long, thin, flattened spindle-shaped cells having thin, dark-staining nuclei. When flat sections or sections parallel to their flat surfaces were made through these cells, they showed elongated, light-staining cytoplasm with generally oval nuclei. The cells, as a rule, presented two to three short processes of the cytoplasm. These cells in every way resembled mature fibroblasts. When the thin lamellae forming the capsular cataract were stained with Mallory's aniline blue or Van Gieson's trinitrophenol-acid fuchsin stain, they were colored blue with the first stain and red with the second. In other words, these lamellae stained exactly the same as any connective tissue.

At the sides or periphery of the capsular cataract the anterior epithelium of the lens presented considerable proliferation of its cells. Just peripheral to the limits of proliferation of the epithelium of the lens the cells of this single layer of epithelium were considerably flattened. The extra amount of flattening here was probably due to the swelling of the fibers of the cortex of the lens accompanying the diffuse cataractous changes. When the epithelial cells became fibroblasts, the cell body, with the nucleus, which was previously of a uniform although attenuated thickness, flattened to a thin disk with pointed edges. All stages in this transition could be studied at the periphery of the capsular cataract. The junction between proliferated epithelial cells and fibrous tissue might or might not be distinct. Posteriorly, behind the fibrous tissue, proliferation of the epithelial cells extended for a short distance toward the sagittal axis of the lens. A layer of epithelial cells lay neither anterior nor posterior to the connective tissue forming the capsular cataract. The latter lay directly on coagulated detritus resulting from liquefaction of the fibers of the cortex of the lens.



Meridional section through one side of the anterior capsular cataract showing proliferation of the epithelial cells of the lens and, to the left, new-formed connective tissue.

CASE 2.—In a boy 7 years old a perforating corneal ulcer due to gonococci was followed by a prominent staphyloma. Enucleation of the eye was performed eight months after the perforation of the ulcer. Anatomically, it was found that the lens had retracted a little after the perforation but was connected with the cicatricial tissue replacing the cornea by a thick column of new-formed connective tissue extending through the pupil and spreading out on the capsule of the lens with a broad base. Under the latter the capsule of the lens was much wrinkled. The capsular cataract was thicker where the connective tissue lay on the outer surface of the capsule, but on one side the cataract extended for a considerable distance with the capsule over it less wrinkled and uncovered by connective tissue. The cortex of the lens generally presented swelling and liquefaction of the fibers.

On the side of the capsular cataract where no connective tissue lay on the surface of the capsule proliferation of cells from the anterior epithelium of the lens could be studied best. The cells in the single layer of lenticular epithelium peripheral to the margin of the capsular cataract were not so flattened as in the

first case. Consequently the difference in thickness between these epithelial cells and their derivatives the fibroblasts was more pronounced. In a fairly uniform manner the proliferated cells from the epithelium of the lens gradually changed to fibroblasts as one followed them toward the sagittal axis of the lens. In some sections proliferation of the epithelial cells had produced cytoplasmic masses with from two to several nuclei. As in the first case, this proliferation of epithelial cells proceeded behind the capsular cataract for a short distance toward the sagittal axis of the lens. On the opposite side of the capsular cataract, where the much wrinkled capsule was covered by a thick mass of new-formed connective tissue, the same change from epithelial cells to fibroblasts was going on, but here the intermediary cells were not so clearly identified. The thickest part of the capsular cataract lying under the thick deposit of connective tissue on the outer surface of the capsule presented in its anterior two thirds very thick lamellae swollen and in part disintegrated; a few flattened cells were scattered here between the layers. All the posterior lamellae of the thicker part, as well as the lamellae of the thinner part of the capsular cataract under the naked capsule of the lens, were thin, with numerous interlamellar cells. The layers of the capsular cataract were all arranged frontally or parallel to the anterior surface of the lens. A layer of epithelium was absent from either in front of or behind the laminated structure. All the lamellae colored blue with Mallory's aniline blue and red with Van Gieson's stain, which definitely indicated their composition of connective tissue. The interlamellar cells had the flattened disk-shaped appearance with processes characteristic of fibroblasts.

COMMENT

In neither case were there encountered the young forms of fibroblasts having vesicular rod-shaped nuclei and rounded ends, which are seen commonly in granulation tissue. A union of the processes from adjacent fibroblasts could be observed in the second case in flat or frontal sections through the capsular cataract. In the first case the cell bodies did not stain uniformly well enough in the flat sections to determine anything regarding this syncytial condition. The surface view of the fibroblasts in both cases showed the nuclei to be generally oval, light-staining and having one nucleolus. With Verhoeff's stain for elastic tissue, elastic tissue proved to be entirely absent in the connective tissue of the capsular cataract.

Other examples could be described, but to do so would simply be to repeat what has already been said regarding the two cases cited. In cases of capsular cataract that are of long standing, however, all connection with, or evidence of, proliferation in the epithelial cells of the lens has usually departed.

SUMMARY AND CONCLUSIONS

The clinical, anatomic and histologic observations in two cases of recent anterior capsular cataract are described. At the periphery of the capsular cataract in each case reported, proliferation of the cells of the anterior epithelium of the lens, with transition of these proliferated cells

to typical fibroblasts, was observed. Observations confirming the fact that the capsular cataract was composed of collagenous connective tissue are offered.

Since the epithelium of the lens is pure ectodermal epithelium in origin, the formation of connective tissue in capsular cataract is an example of true metaplasia. The designation of capsular cataract is therefore well taken, since the capsular cataract, like the capsule of the lens (Seefelder⁹), is formed from the epithelium of the lens.

9. Seefelder, R., in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 1, p. 499.

THE GOLD BALL IMPLANT

SOME ESSENTIAL FEATURES IN OPERATIVE TECHNIC

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The use of implants after enucleation has occupied the attention of ophthalmic surgeons for more than fifty years. A variety of substances have been tried, such as fat, cartilage, bone, ivory, celluloid, paraffin, peat and agar-agar. With the discovery of its shortcomings, each of these has been successively discarded. The material of choice is now a gold or glass ball. The gold ball is generally regarded as the preferred implant. Though in this article I shall discuss the gold ball implant, the technic described applies equally to any form or variety of implant.

In my early experience with implants I paid as little attention to the details of the technic of implantation as does the average writer on the subject. My efforts were crowned with failure and escape of the implant in about half of the cases. These disappointing results led me to study carefully the part played in the unsuccessful attempts by various factors in the technic of the operation.

The first deleterious factor in technic was found to be the use of too large a ball. At the suggestion of Dr. Arthur Chambers, I began to use a sphere not greater than 14 mm. in diameter. Such a sphere was found to fill Tenon's capsule without causing undue tension on the delicate capsular tissue. The successful results increased in number following this procedure.

It was then found that failure to control meticulously any oozing of blood within Tenon's capsule was directly responsible for some failures. Attention paid to this phase of the operation further increased the percentage of successes.

The manner in which Tenon's capsule was sutured was then considered. It appeared that retention of the implant would possibly be better if the edges of Tenon's capsule were overlapped rather than if the margins were merely approximated and sutured with interrupted sutures. The successes following overlapping of the margins proved that this factor in technic was of high significance.

It is impossible to conceive of any success attained by merely suturing the conjunctiva without regard to Tenon's capsule as described by some authors. It is absolutely essential that Tenon's capsule be separately and carefully sutured over the implant.

My present technic has given consistent retention of the gold ball in the last ten cases over a period of five months. The procedure is as follows:

For local anesthesia, a few drops of a 4 per cent solution of cocaine hydrochloride is placed within the conjunctival sac. The usual circumcorneal incision is made. To conserve all the conjunctival tissue possible and to have a cleancut incision, I first pass the point of a knife around the limbus, as suggested by Dr. Edwin Munson, and proceed to undermine the conjunctiva back to the equator. At this stage I inject not more than 1 cc. of a 4 per cent solution of procaine hydrochloride, directing the needle along the outer orbital wall for a distance of 3 cm., with the point directed slightly upward. Here the three branches of the ophthalmic division of the trigeminal nerve enter the orbit through the sphenoidal fissure. These branches and the ciliary ganglion are blocked; this gives sufficient anesthesia for the entire operation.

Each of the four rectus muscles is successively caught on muscle forceps and sutured with 000 catgut to the conjunctiva. Double-armed sutures are passed from the under surface of the muscle through the conjunctiva about 5 mm. from the cut margin. This allows the muscles to assume approximately the same position as in the living eye. The ends of the catgut sutures are left long, held by an artery forceps and laid to one side. The globe is proptosed and cut away with as little loss of tissue as possible. A large spindle-shaped mass of cotton made moist with hot saline solution is packed into the socket until hemorrhage is completely controlled. A dry packing is placed in the socket and kept there to dry the cavity further while one proceeds with the suturing of the edges of Tenon's capsule.

One margin of Tenon's capsule is grasped with a tissue forceps, and a double-armed 000 chromatinized catgut suture is introduced from below at a point 2 mm. back of the center of the cut margin of the capsule. This suture is carried to the opposite margin and introduced from below in a similar manner. The suture is left untied for the moment. Two or three additional sutures placed on each side of this one at intervals of 2 mm. are similarly dealt with. The packing is now removed, and a dry cavity will be found. The gold ball is put into place, the sutures are tied, the center one first and then those on each side. The margins of the capsule overlap. If additional sutures are required to close the cavity completely, they can be introduced. A running catgut suture may then be placed in the capsule for reenforcement. The conjunctival wound is closed with no. 2 braided silk.

The four catgut sutures, each of which fixed a rectus muscle to the conjunctiva and was laid to one side at the outset of the operation,

are brought in apposition and loosely tied. Insertion of petrolatum or, preferably, ointment of scarlet red, into the conjunctival space, and a dressing complete the operation. The silk sutures are removed after one week, and at this time the superficial catgut sutures are easily picked off.

My experience indicates that four features in the technic of implantation of the gold ball are essential for success.

1. Tenon's capsule must be sutured separately over the implant. It seems needless to stress this point were it not for the fact that it is omitted by some operators.

2. Oozing of blood in the cavity of the capsules must be carefully controlled.

3. The ball must be small, not greater than 14 mm. in diameter.

4. Closure of Tenon's capsule by overlapping of the margins is essential.

CORALLIFORM CATARACT AND A NEW FORM OF CONGENITAL CATARACT WITH CRYSTALS IN THE LENS

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AND

IRVING PUNTENNEY, M.D.

CHICAGO

The name coralliform cataract was first applied by Gunn¹ to a particular type of congenital cataract in which many layers of the lens in the axial area are affected by peculiar branching opacities. Related forms of opacity which have this peculiarity of distribution have been called floriform, arborescent and fusiform cataract, while the form described by Vogt² as spear cataract also belongs in this group. In the latter's cases the opacities were composed of shiny crystals arranged about denser central rods to form a series of spear-shaped figures joined together at irregular angles more or less near the sagittal axis of the lens. The opacities gave the impression of a number of spiny insects or caterpillars in the axial area. A mother, a son, a maternal aunt and two cousins were affected. In 1924 one of us³ reported a case resembling that of Vogt in which similar crystals were arranged more regularly in the axial area so as to resemble two fir trees with their bases together in the center of the lens, the tips coming nearly to the anterior and the posterior capsule. The only other cases of a condition designated by the name spear cataract which we have found in the literature are those of two sisters, reported by Cords,⁴ though the relation between this form of opacity and the forms described by other names is so close that sharp differentiation is in many cases impossible. In 1927 one of us⁵ reported two cases of the more common form, which could be described as coralliform. The literature on the few reported cases

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1. Gunn, R. M.: *Tr. Ophth. Soc. U. Kingdom* **15**:119, 1895.

2. Vogt, A.: (a) *Arch. f. Ophth.* **107**:446, 1922; (b) *Lehrbuch und Atlas der Spaltlampen Mikroskopie des lebendes Auges*, Berlin, Julius Springer, 1931, pt. 2, p. 446.

3. Gifford, S. R.: *Am. J. Ophth.* **7**:678, 1924.

4. Cords, R.: *Klin. Monatsbl. f. Augenh.* **76**:125, 1926.

5. Gifford, S. R.: *Klin. Monatsbl. f. Augenh.* **78**:194, 1927.

in these groups will be found in these papers. In nearly all the cases the opacities were bilateral, and often several members of a family were affected. In the family of Gunn's patient Nettleship found nineteen cases of typical coralliform opacities. In the family studied by Knies, anterior and posterior polar cataracts connected by a threadlike opacity to a lamellar opacity near the center of the lens were present in two members, while two other members showed anterior polar and nuclear opacities.

Apparently in the whole group of cases the lesion first appears in early fetal life, affecting the region of the embryonal nucleus. Layers of cortex of later development are each affected in the central area only, the extent of opacity being often less in the succeeding layers till, as in my cases, a layer next to the capsule may be completely spared.

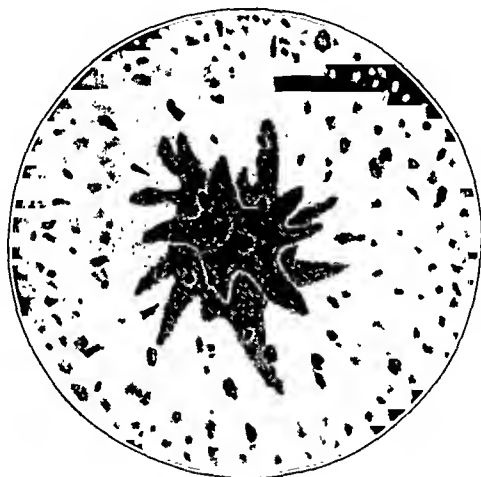


Fig. 1.—Opacities in the lens of the right eye in case 1.

We are reporting an additional case of coralliform cataract and also a case of congenital cataract of even more unusual form, observed within the past three years, in which it was possible to investigate the character of the lenticular opacities by microchemical methods. The investigation was fairly complete in case 2 only, because of insufficient material in case 1.

REPORT OF CASES

CASE 1.—Mrs. J., a woman of 28, had first noticed poor vision at the age of 18. Vision rapidly became worse, so that four years later she was unable to read. There was a history of cataract in the family. The mother was told at 32 that she had "blue cataract." When she was seen by one of us at the age of 50, however, the cataracts were far advanced and could not be distinguished from senile cortical cataract. In a maternal uncle cataract developed at the age of 55. The patient has two older sisters and one brother who know of no ocular defect.

When the patient was first seen vision was 4/200 in the right eye and 20/100 in the left. The right eye showed opacities of the lens as illustrated in figure 1.

The irregular stellate or flower-shaped opacities extended forward to the anterior capsule and backward to the posterior capsule, being connected with similar branching opacities involving all layers of the lens in the axial area. Outside this area the cortex contained many fine punctate and larger flaky opacities; as seen with the slit lamp these gave a play of colors from green to purple. The left eye showed a similar picture except that the branching axial opacities were not so extensive. The opacity in the anterior capsule was limited to a small oval area.

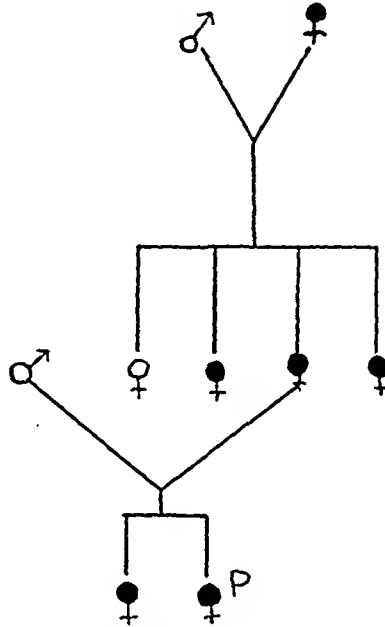


Fig. 2.—Family tree of the patient in case 2. The black symbols indicate affected patients.

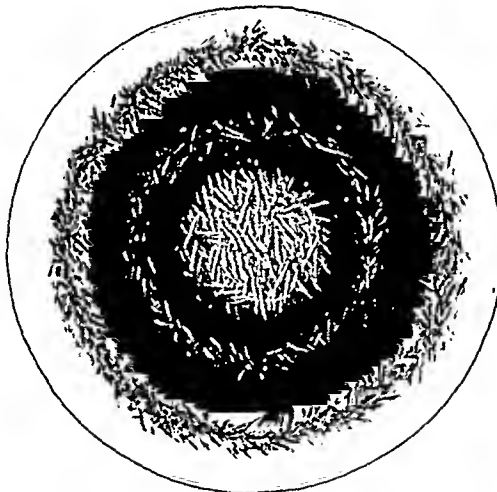


Fig. 3.—Opacities in the lens of the left eye in case 2.

Within a period of six months both lenses were removed by corneal expression after preliminary discission, with resulting vision of 20/15 in each eye. The expressed material was collected and examined microscopically. The fresh moist material showed long needle-shaped crystals somewhat resembling those seen in case 2, but no photomicrograph was taken. The crystals were insoluble in chloroform and a 70 per cent solution of alcohol. They were at least relatively insoluble in

cold water, since their shape was preserved in moist preparations. Acids and alkalis were not tried, nor were any chemical determinations made. Insolubility of the crystals in chloroform, ether and alcohol apparently excludes cholesterol and other lipoids, so far as the larger crystals are concerned. Millon's reaction was negative, so it was thought that the protein crystals described by Verhoeff could be excluded.

CASE 2.—Miss V., aged 20, whose vision had been poor since infancy, had undergone needling of the right eye at the age of 13. Owing to the death of the operator

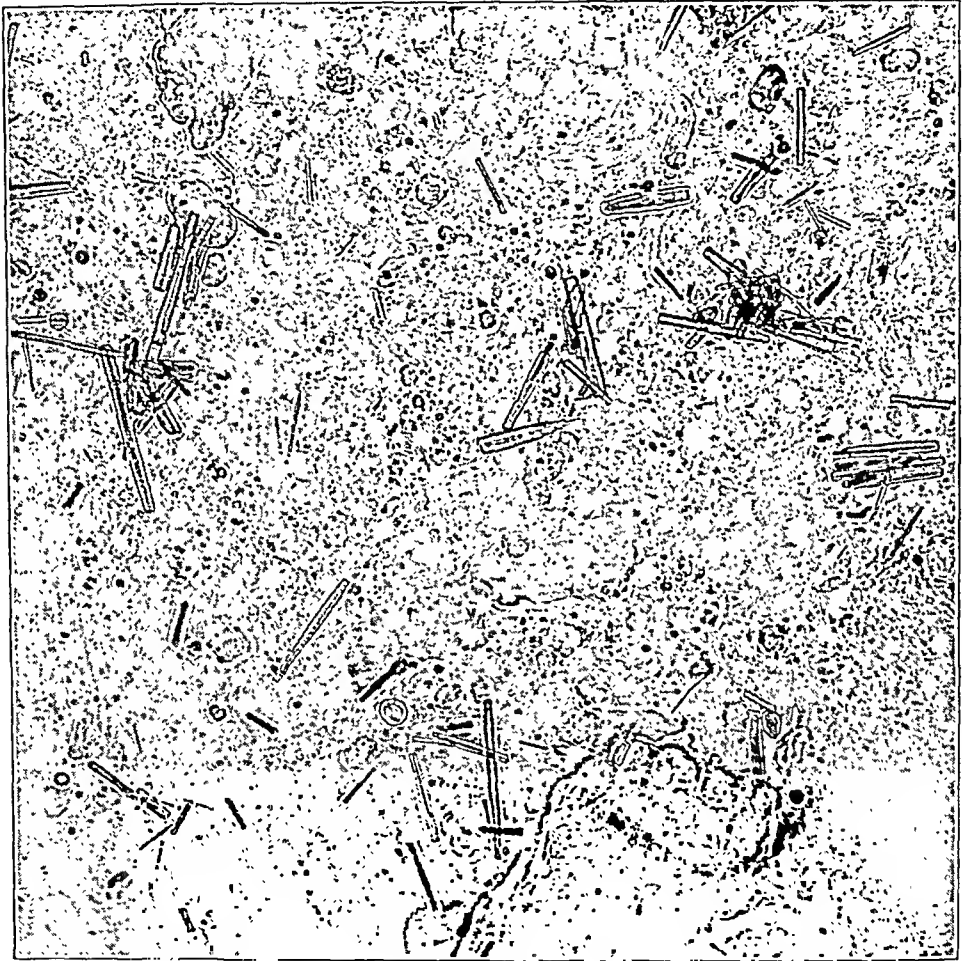


Fig. 4.—Photomicrograph of crystals of the opacity in case 2.

not long afterward, no subsequent operations were done, and she had attended the Indiana School for the Blind.

The patient knew that congenital cataract was common in the maternal side of her family, the mother, maternal grandmother and four maternal aunts being affected, while one maternal aunt was spared. The patient's only sister was affected (fig. 2). The patient had no brothers. The two maternal aunts had previously been cared for by Dr. H. Gifford and one of us. One aunt at the age of 18 months showed what were described as zonular cataracts with several distinct yellowish spots in the nuclei. Vision of 20/70, as found fifteen years later,

was obtained after discissions. The other maternal aunt, who was seen at the age of 37, showed "very dense lamellar cataracts, about 6 mm. in diameter." No mention of crystals is made in the record. Vision of 20/25 and 20/30 was obtained after discission and corneal expression.

Vision of the right eye of our patient was 15/70, and that of the left eye was 20/100, with correction. The right eye showed a fairly dense membrane, with a small opening and apparently no cortex remaining. The left eye showed the condition illustrated in figure 3. The opacity was composed of crystals which resembled those seen in the case of spear cataract but were arranged in a more compact and circular mass. This mass could be divided into an outer zone measuring 6 mm. in diameter and an inner one measuring 3 mm. The space between these zones

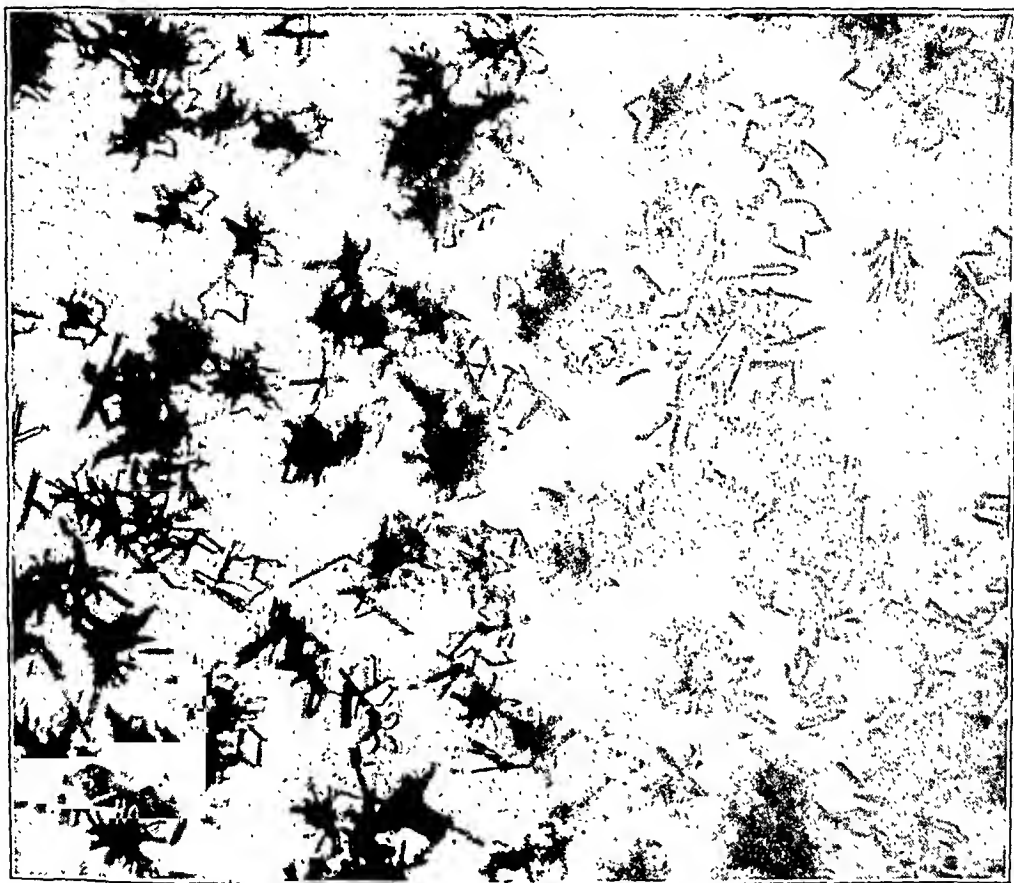


Fig. 5.—Photomicrograph of crystals of calcium sulfate.

also contained crystals, but these were more sparsely distributed. The small inner mass was localized by the anterior Y suture which rested on its anterior surface. A thin layer of normal cortex was present anterior to the outer zone of crystals. Presumably it extended to the posterior cortex, but this could not be seen. The crystals were needle shaped and of various sizes. They were not so shiny as those seen in case 1, and no play of color was observed.

Obviously this picture did not resemble that of case 1, nor was it like that of Vogt's spear cataract. While the concentric zones of denser opacity suggested a development similar to that of zonular cataract with involvement of successive layers of cortex, the fact that the whole opacity consisted of crystals distinguished the picture definitely from that of ordinary zonular cataract. The arrangement can perhaps best be compared with the zones of light seen in a revolving pinwheel.

I have not found descriptions of its exact counterpart in the literature. In the cases described by Vogt⁶ the eyes presented some points in common, especially in one case, in which there were "rosette-like concentric club-shaped opacities," but in none was there any close resemblance.

In the right eye a cut through the membrane by Wheeler's method produced an opening through which vision of 20/40 was obtained with correction. In the left eye a preliminary dissection was done, followed in three days by corneal expression of the soft cortex. The central mass of opacities remained in a fairly coherent isolated mass and was collected in a large pipet for immediate examination. Some cortex remained which absorbed slowly; vision was 20/50 three months after operation.

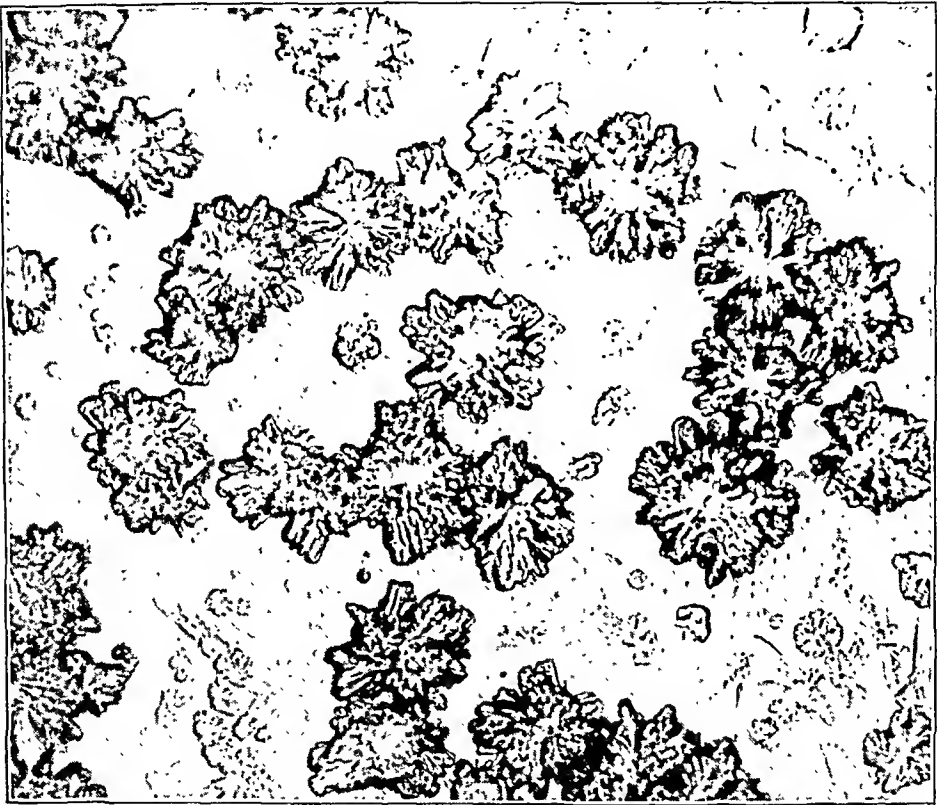


Fig. 6.—Photomicrograph of crystals of calcium phosphate.

Examination of the soft material containing the opacity showed the crystals illustrated in figure 4. These were long polyhedral needles of fairly regular width but varying in length. They resembled most closely the needle-like crystals of calcium sulfate (fig. 5). Some crystals of calcium phosphate are shown in figure 6 for comparison.

The crystals were insoluble in alcohol, ether, glycerin, dilute ammonium hydrate and hot and cold water. They were soluble in dilute sulfuric acid. Insolubility in ether excluded lipids and cholesterol as constituents of the crystals. They

6. Vogt,^{2b} pp. 429, 433 and 444.

resembled alanine crystals in form, but this and other amino-acids were excluded because of insolubility in hot and cold water. Solubility in dilute sulfuric acid made it appear almost certain that they contained a calcium compound.

In view of this the remaining material was employed for a quantitative determination of calcium. This showed 0.6 per cent of calcium in the moist material.

COMMENT

According to Salit,⁷ the normal lens contains no calcium, while Adams⁸ found 0.0185 per cent. In lenses with incipient cataract Salit found 0.0058 per cent, and in those with mature senile cataract, 0.0509 per cent, the greatest amount in any lens being 0.108 per cent. Adams found 0.0653 per cent in lenses with cataracts of various types. Hence the amount of calcium found in the opaque portion of our lens was ten times the average amount found in cataractous lenses by Adams, which offers additional proof that the crystals examined were composed chiefly of calcium.

The presence of crystals in the cataractous lens is not uncommon. In the cataract of patients with myotonic dystrophy crystals are nearly always observed and from their flaky form and the play of colors observed with the slit lamp have usually been considered as cholesterol crystals. A few recent reports by Cattaneo,⁹ Kranz¹⁰ and others have shown that by means of examination by polarized light the presence of cholesterol, other lipids and calcium may be determined in the cataractous lens before operation. Kranz found that 30 per cent of normal lenses showed some crystals, chiefly lipid crystals, by this method, while in cases of the common coronary cataract crystals were found in 80 per cent. He did not examine any lenses resembling those here described.

Cases of congenital cataract showing crystals which have been examined microchemically are few in number. Braun¹¹ examined with especial care the lenses of a woman with cataract greatly resembling the coralliform type. A son was affected with identical opacities in the otherwise clear lens. Braun found the crystals to be those of a compound of tyrosine. Calcium and cystine were also found but were not considered as principal constituents of the crystals. Vogt found in one of his cases of spear cataract crystals which consisted chiefly of cysteine, though cholesterol could not be excluded. Beresinskaja¹² described

7. Salit, P. W.: *Am. J. Ophth.* **13**:1072, 1930; Calcium Content and Weight of Human Cataractous Lenses, *Arch. Ophth.* **9**:571 (April) 1933.

8. Adams, D. R.: *Biochem. J.* **23**:902, 1929.

9. Cattaneo, D.: *Ann. d'ocul.* **165**:105, 1928.

10. Kranz, H. W.: *Klin. Monatsbl. f. Augenh.* **79**:654, 1927; *Ztschr. f. Augenh.* **65**:219, 1928; *Arch. f. Ophth.* **118**:571, 1927.

11. Braun, G.: *Arch. f. Ophth.* **118**:701, 1927.

12. Beresinskaja: *Klin. Monatsbl. f. Augenh.* **80**:125, 1928.

a familial zonular cataract in which crystals could be seen which proved on analysis to contain cholesterol and some traces of cystine. Verhoeff¹³ examined carefully the lenses of a man with typical coralliform cataract. The crystals were larger than those seen in our cases and were insoluble in acids, alkalis, alcohol and ether. They were digested by pepsin in a 0.4 per cent solution of hydrochloric acid and gave a positive test with Millon's reagent. They were considered to be a crystalline form of protein like that found in certain plants.

CONCLUSIONS

The reports indicate that various chemical changes may occur in lenses with opacities belonging in the group of coralliform cataract, resulting in the formation of crystals in the lens.

In certain cases the crystals themselves seemed to be chiefly responsible for the opacity. Thus, in the cases studied by Vogt, Verhoeff and Braun and in the second case described here the lenses were clear except for the crystals.

While resembling each other in distribution and in general appearance, on careful slit lamp study the crystals revealed in most cases differences which, in view of the chemical findings in the cases studied, would give some indication of their chemical nature. One is not justified in calling any crystals that give a play of colors cholesterol crystals.

There seems to be no doubt that the crystals in our second case were those of a salt of calcium, probably calcium sulfate, while in the first case it is probable that they were of a similar nature.

That a chemical process which results in the formation of such crystals in the lens should be transmitted by heredity is noteworthy. This is not true in all cases, since in certain families other forms of cataract, not characterized by crystals, occurred. It was noteworthy, however, in the families studied by Vogt and Braun in which identical crystals and no other changes were present in two generations.

13. Verhoeff, F. H.: *Arch. Ophth.* **47**:558, 1918.

Clinical Notes

AN AID TO FACILITATE THE OPHTHALMOSCOPIC EXAMINATION

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The ophthalmoscopic examination is made with less effort and therefore with less fatigue on the part of the patient if it is possible to orient oneself immediately. When the eye is so placed that the ophthalmologist looks straight through the pupil at the optic nerve head he can quickly make his study. The purpose of the procedure that I use is to enable the patient to place his eye in the position of choice and to make it easier for him to keep his eye in this position.

One encounters little difficulty in making the ophthalmoscopic examination of many patients when using the routine directions, namely, asking the patient to look straight ahead, to look slightly to the right or to the left, to look at the ceiling or to look at some object. However, the patient may find difficulty in following such directions, especially when the examiner's head necessarily obstructs his view in part or completely. It has been my experience that when the vision of the patient is obstructed by my ophthalmoscope and my head his eye will often wander. The procedure to prevent this has been evolved. I attempt to utilize the patient's sense of position so as to place the eye in the optimal position for ophthalmoscopic examination. In the case of babies I attempt to utilize their sense of hearing and position, and in the case of psychotic persons, to utilize their sense of hearing, their sense of position and their sense of feeling.

The patient is instructed to place his right arm straight out at right angles to the chest and is told to look at his right thumb. (It is usually easier to take the patient's hand and place it in the desired position and tell him to hold it there and look in the direction of his thumb.) The ophthalmologist's head may prevent the patient from seeing his thumb, but he knows where his thumb is and can focus his eye in that direction whether or not he can see it. When the right thumb has been placed forward, straight out from the shoulder and at right angles to the chest, it is in perfect position to place the left eye in the best location for examining the fundus, and it follows that the left thumb so placed fixes the right eye in the correct location for examination. The advantage to the patient, particularly to a nervous patient, is definite when one is able to cut down the time necessary to make the ophthalmoscopic examination. The student who is able to look straight through the pupil at the disk is encouraged to look at more eyes, and the patients who are used for demonstration do not become fatigued as quickly when the time required for this study by the student is markedly reduced.

From the Department of Neuropsychiatry, the Medical College of Virginia.

Certain patients—those with paresis, paralysis, disturbances of consciousness of varying degrees or involvement of attention to various extents—are unable to hold their hands in the correct position; this procedure is of much value in such cases. In these cases I have the nurse hold the patient's hand and ask him to look at his thumb, augmenting his attention by placing in his hand something warm or something cold. This will tend to make the patients place their eyes in the proper position, and though they are mentally disturbed my associates and I have been able to reach their consciousness to the extent of getting brief periods of cooperative effort on their part.

The examination of children is facilitated occasionally by having the nurse hold in their hands a watch or a toy, preferably a toy that will make a noise, such as a small crying doll. In the case of young babies I place a watch by the side of the baby's ear, let him look at the watch, place it back by the side of his ear and then have the nurse hold the watch in his hand, which is held in the correct position. In this way I utilize the patient's attention to his hand, to the toy, to the watch, and to the sound made by the watch or the toy.

The location of the object in the hand when the arm is held at right angles to the chest on a level with the shoulder, or slightly higher, as the examiner prefers, enables the nurse to keep the objects used for attention anchored in the optimal position to fix the eye so that the examination is made with the greatest of ease.

I do not know whether this method has ever been used before or whether it is in general use by some physicians. It is a procedure that I have evolved and found workable for my students and myself.

INSTRUMENTS FOR TREATMENT OF THE LID

PARKER HEATH, M.D., DETROIT

Herewith is submitted a set of instruments for treatment of the lids which save a great deal of time for the surgeon. The instruments are shown in the accompanying illustrations. They are made by V. Mueller and Company.



Fig. 1.—A dilator for the punctum, of convenient size and with a nonslipping handle, which can be used with a sharp needle point or with a duller point.



Fig. 2.—A double-ended instrument for massage and expression of the contents of the meibomian and other glands of the lids.



Fig. 3.—A convenient conjunctival flap dissector, useful especially in operations with the trephine, which enables one to dissect out a pocket between the scleral ingrowths, the favorite site for making a trephine opening.



Fig. 4.—A series of ring curets, with dull and sharp sides, which are easily kept clean and are easy to manipulate, especially in operations for chalazion.

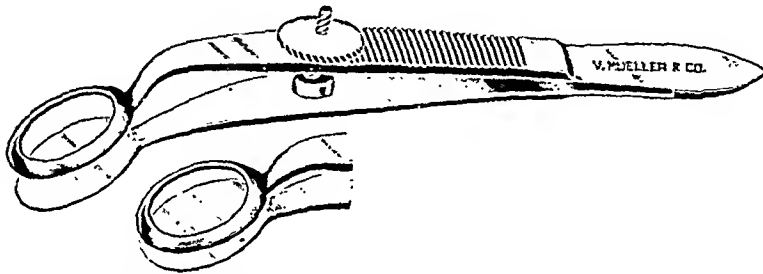


Fig. 5.—A chalazion clamp of the proper shape, one that will not compress the border of the lid.

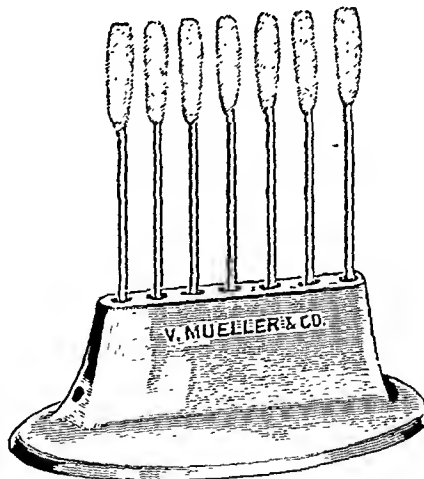


Fig. 6.—An applicator holder which is easily kept clean and which enables the surgeon to select one applicator easily without contaminating the others.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

EARLY, SIMPLE GLAUCOMA

ITS DIAGNOSIS AND MANAGEMENT

FREDERICK C. CORDES, M.D.

SAN FRANCISCO

Glaucoma is a collective term applied to all diseases of the eye the principal symptom of which is increase in the intra-ocular pressure. In certain eyes the intra-ocular tension is above the average normal but causes no functional changes; this condition cannot be called glaucoma.

NORMAL INTRA-OCULAR TENSION

The tension of the normal eye in the vast majority of cases varies between 21 and 27 mm. of mercury. According to P. Knapp,¹ Langenhan² and Bruns,³ this is uninfluenced by sex or refraction, while Guglianetti⁴ found that temperature, humidity and atmospheric pressure did not play a rôle.

The intra-ocular pressure is not absolutely constant but shows certain slight variations. The influence of the variations of the blood pressure due to the respiratory rate and the pulse rate, as expressed by the rhythmic oscillations of the pressure, can be seen roughly by the movement of the pointer on the Schiötz tonometer. A second form of deviation is the regular daily variations. Regular tonometer readings over twenty-four hour periods show a daily variation of from 2 to 3 mm., the pressure being higher in the morning than at night. Many causes have been suggested for this, such as the horizontal position in sleep, the absorption of food, failure of the pumping action of accommodation and the absence of muscular movement.

Szymanski and Wladyczko⁵ were able to demonstrate experimentally that an excavation of the disk follows a rapid decrease of intracranial

From the Division of Ophthalmology, the University of California Medical School.

1. Knapp, P.: *Klin. Monatsbl. f. Augenh.* (pt. 1) **50**:691, 1912.

2. Langenhan, in von Graefe, A., and Saemisch, E. T.: *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1904, vol. 4, p. 606.

3. Bruns: *Klin. Monatsbl. f. Augenh.* **71**:90, 1923.

4. Guglianetti, L.: *Arch. di ottal.* **21**:382, 1913-1914

5. Szymanski and Wladyczko: *Klin. Oczna* **3**:145, 1925; abstr., *Zentralbl. f. d. ges. Ophth.* **16**:729, 1926.

pressure, while Parker⁶ was able to produce choked disks by rapid reduction of the intra-ocular tension. Block and Oppenheimer,⁷ however, found that there is no apparent relationship between the intra-cranial pressure and the intra-ocular tension, although both are affected by a change in the osmotic concentration of the blood.

A good deal has recently been written on the relationship of the intra-ocular pressure and the blood pressure, the permeability of the vessels and its effect on the intra-ocular tension. While this work is in the theoretical stage, it seems to be pretty well established that a sudden large variation of the general blood pressure can produce changes in the intra-ocular pressure.

The effect of the glands of internal secretion on the vascular system is well known. That these glands also affect the intra-ocular tension has been brought out by experimentation and clinical observation. Hertel⁸ found that extirpation of the thyroid caused an increase of the intra-ocular tension, while feeding thyroid produced a drop in the pressure. These observations were confirmed by A. Fuchs,⁹ Lamb¹⁰ and others. Imre¹¹ observed that in hyperpituitarism the tension was rather low and that pregnant women showed the tendency to low tension. Cucchia¹² found that extirpation of the ovaries had no influence on the tension, while Salvati¹³ found a slight increase in the tension during menstruation. Such observations as the aforementioned have led to attempted treatment of glaucoma by organotherapy. From the clinical and experimental observations it seems evident, however, that the endocrine glands do influence tension.

The relation of the intra-ocular tension and nervous influence has long been recognized. Research and clinical observations, such as those of Abadie,¹⁴ have shown that the regulation of the intra-ocular tension is a function of the sympathetic nervous system. Henderson and Starling¹⁵ in 1904 stated that the intra-ocular pressure is a function of the blood pressure in the ocular blood vessels and varies directly as the latter. They demonstrated that the sympathetic nervous system contains

6. Parker, W.: *Tr. Am. Acad. Ophth.* **29**:77, 1924.

7. Block, E. B., and Oppenheimer, R. H.: *A Comparative Study of Intra-spinal Pressure, Blood Pressure and Intra-Ocular Tension*, *Arch. Neurol. & Psychiat.* **11**:444 (April) 1924.

8. Hertel, E.: *Arch. f. Ophth.* **88**:197, 1914.

9. Fuchs, A.: *Bull. Ophth. Soc. Egypt.*, 1924, p. 23.

10. Lamb: *Tr. Am. Ophth. Soc.* **24**:105, 1926.

11. Imre, J., Jr.: *Endocrinology* **6**:213, 1922.

12. Cucchia, A.: *Ann. di ottal. e clin. ocul.* **56**:117, 1928.

13. Salvati: *Ann. d'ocul.* **160**:568, 1923.

14. Abadie: *Clin. opht.* **12**:303, 1923.

15. Henderson, E. E., and Starling, E. H.: *J. Physiol.* **31**:305, 1904.

vasoconstrictor fibers going to the uveal blood vessels and that stimulation causes constriction of the intra-ocular blood vessels. Adler, Landis and Jackson¹⁶ were able to show experimentally that an increase of the blood pressure produces an increase in the intra-ocular tension. The local vasoconstriction through the cervical portion of the sympathetic nervous system, however, acts as a protective measure to prevent sudden harmful increase in the intra-ocular pressure. These authors also found no effect on the intra-ocular tension when the cervical portion of the sympathetic trunk was cut when the blood pressure was normal. After section of the sympathetic trunk, however, there was a higher intra-ocular pressure than in the control animal with an increased blood pressure. Stimulation of the cervical sympathetic nerve fibers shows, as a rule, a temporary reduction of tension in both normal and glaucomatous eyes.

From the brief review of the physiologic features of the intra-ocular tension just given it becomes apparent that many factors have a bearing on its control. This explains the difficulty in understanding the various elements that play a part in the production of primary glaucoma. It is possible that in the future ophthalmologists may have to change completely their conception of glaucoma and may perhaps be able to classify it on an etiologic basis.

GLAUCOMA

At the present time glaucoma, exclusive of the congenital type (hydrophthalmos), is divided clinically into two main groups, primary and secondary. The cases in which glaucoma results from visible pathologic changes in the eye, such as occlusion of the pupil, are known as cases of secondary glaucoma. In contradistinction to secondary glaucoma, there is primary glaucoma. In this type there is no demonstrable disease of the eye that can be regarded as a causative factor.

Primary glaucoma is not a single symptom complex. The different forms are determined by the height of the intra-ocular pressure, the localization and degree of the pathologico-anatomic changes and the resulting disturbance of function. The variation in the picture of the symptom complex caused by these factors suggests a classification of primary glaucoma into the following types: (1) acute inflammatory (acute uncompensated), (2) chronic simple (compensated), (3) chronic inflammatory (chronic uncompensated) and (4) absolute (degenerative). Here I am concerned only with chronic simple glaucoma in its early stages.

16. Adler, F. H.; Landis, E. M., and Jackson, C. L.: *Arch. Ophth.* **53**:230, 1924.

In glaucoma simplex both eyes are usually involved, and the intra-ocular pressure is, as a rule, consistently higher than in the normal eye. While these eyes show a complete regulation of the production and drainage of aqueous, the fine balance of the pressure-regulating apparatus has been disturbed. A certain balance, however, still persists, so that acute increase of pressure is not present and thus there is no dilatation of the veins. It is because of this lack of congestion that simple glaucoma is also called compensated glaucoma.

In the beginning of the disease the subjective signs are mild or completely absent. Pain, foggy vision and rainbow colors are, as a rule, absent. The persistently increased intra-ocular tension gradually produces excavation and atrophy of the optic nerve. It is only when these factors produce disturbance of vision that the patient seeks medical aid. If the change is gradual the condition may not be observed or may be attributed to senile changes. If one eye is involved the condition may be called to the patient's attention only when some accident makes him close the other eye. Externally the eye cannot be differentiated from the normal eye except by certain detailed minute examinations. The ophthalmoscopic examination and the manual palpation of the eye during a routine examination may reveal signs that lead one to suspect early glaucoma. As Gradle¹⁷ has pointed out, there are also certain hints that may be derived from the history. In a patient in the age period when glaucoma is most prevalent "the story of headaches appearing after a stay of twenty minutes or more in a darkened room, such as at a moving picture theatre, should arouse suspicion. Again, headaches appearing during the morning hours may demand further investigation. In some instances, a digestive upset is the precursor of a simple glaucoma." The presence of glaucoma in the family history should also make one suspicious.

The early diagnosis of simple glaucoma is of great importance and is often difficult. The borderline cases are all too frequently overlooked, and it is for the detection of these cases that the ophthalmologist must be ever on the alert. In cases in which the condition is suspected, in addition to the routine examination a study of the curve for tension and certain provocative tests for compensated glaucoma are indicated. These tests often point to the diagnosis and give the indication for the type of therapy demanded.

INTRA-OCULAR TENSION IN GLAUCOMA

The height of the intra-ocular tension in glaucoma varies a good deal. Most frequently the tension is between 30 and 50 mm. of mercury. There may be instances in which the pressure is higher or lower. There are, on rare occasions, cases in which the pressure is below the upper

17. Gradle, H.: *Am. J. Ophth.* **14**:936, 1931.

limits of normal and in which all the signs of glaucoma are present. These are referred to as cases of "glaucoma without increased tension."

In these cases of glaucoma without increased tension, according to A. Knapp,¹⁸ the condition is not glaucoma. While the patient shows atrophy of the optic nerve with excavation of the disk, he does not show increased tension at any time. The perimetric changes have a tendency toward altitudinal defects and do not correspond to the usual defects of the field seen in glaucoma. There is also a tendency to show no progression after a certain stage. The tension is often low. In one of the three cases that I have personally observed the tension never rose above 18 mm. (Schiötz) even when the tension was taken over the twenty-four hour period. Roentgen examination shows calcification of the internal carotid, posterior communicating and ophthalmic arteries, which Knapp feels is the etiologic factor.

The change from normal tension to pathologic tension in glaucoma simplex is gradual and insidious. Sudden changes such as are noted in acute glaucoma do not occur. In the course of years the tension gradually and slowly increases until it reaches a pathologic height. There is, however, a regular daily and yearly variation that is important from a diagnostic and a prognostic point of view. In the understanding of glaucoma a curve for tension is just as important as a curve for temperature in infectious diseases.

DAILY CURVE FOR TENSION

If the tension is taken every four hours during the twenty-four hours of a day, the normal eye will show no appreciable variation (fig. 1). In the glaucomatous eye, on the other hand, there is a definite variation, which may amount to 20 mm. of mercury. The intra-ocular tension reaches its highest point between 5 and 7 a. m. On arising there is a rather sudden drop, the tension remaining lower until late at night. At night the rise takes place in two states—a rather gradual increase until 3 o'clock and then a sudden rise that reaches its high point between 5 and 7 o'clock (fig. 2). According to Thiel,¹⁹ this is the typical curve for glaucoma simplex as shown by the examination of extensive clinical material. This curve has been explained as due to change in the distribution of the blood of the body, the cessation of digestion, probably being an important factor. Taken over a period of days the curve for tension shows a consistent daily variation (fig. 3). If both eyes are involved the curves are similar, whereas if only one eye is affected the other will show but little variation. If, however, the apparently normal eye shows a variation of more than 10 mm. this must be regarded as a sign of latent glaucoma.

18. Knapp, A.: Association of Sclerosis of the Cerebral Basal Vessels with Optic Atrophy and Cupping, *Arch. Ophth.* 8:637 (Nov.) 1932.

19. Thiel, R.: *Arch. f. Augenh.* 96:331, 1925.

From this it appears that in cases in which the signs lead one to suspect early glaucoma, a reading of tension once a day is not sufficient, but the tension should be taken twice a day, once in the early morning

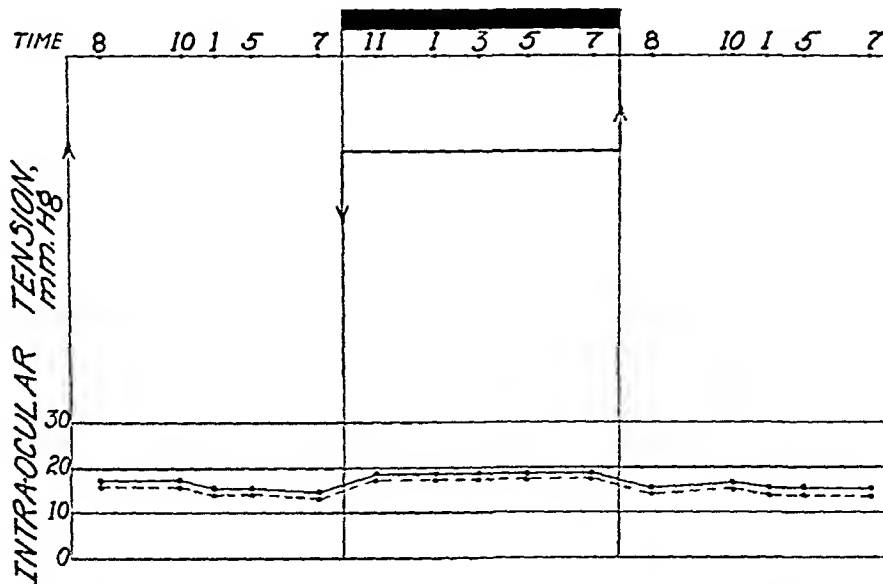


Fig. 1.—Daily curve for tension of a normal right and left eye. Charts 1 to 6 were adapted from Thiel, R., in Schieck, F., and Brückner, A.: *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 4, p. 717.

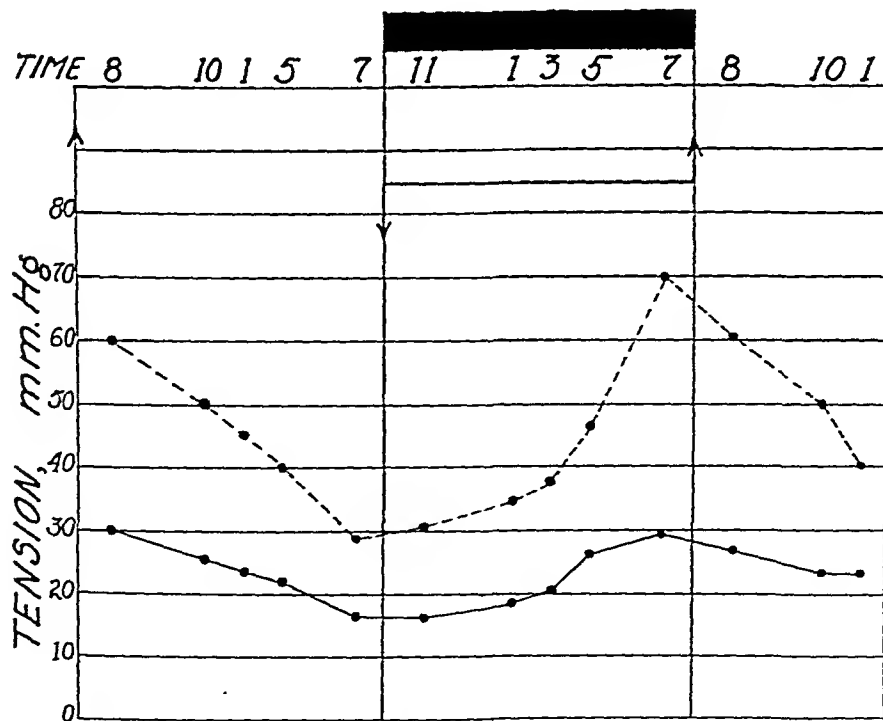


Fig. 2.—Typical curves for tension for two eyes with early chronic simple glaucoma.

and again late in the afternoon. If possible, hospitalization, with taking of the tension during intervals over from three to four days, is preferable. In regard to the early morning tension, it is important to take

this at a time when it is necessary to awaken the patient to take the tension. From a practical point of view it is not necessary to awaken the patient during the early morning hours. Taking the tension every four hours during the day until 11 p.m. and then again at 6 a.m. will suffice to bring out any abnormal curve. In those cases in which the tension remains normal during the day but rises to abnormal heights during the night, the curve for tension is particularly valuable and will explain the presence of early changes in the central field.

If the curve does not show the typical form or if hospitalization is not possible, it is often possible to establish the diagnosis by the so-called provocative tests. The adaptability of the eyes to appreciable variations in the blood pressure and the changes in the distribution of blood form the basis for these tests. While the intra-ocular vascular system

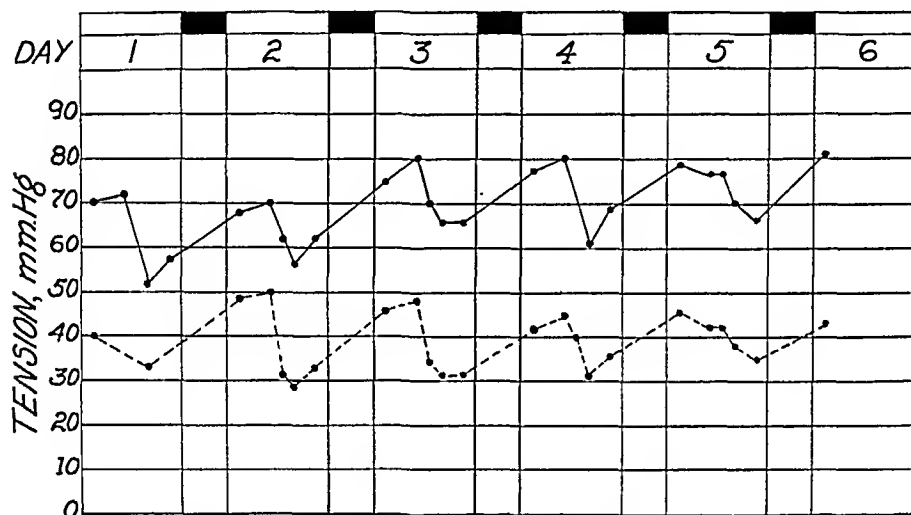


Fig. 3.—Constant daily curve in glaucoma simplex for the right and the left eye.

shows these changes, the tension is unaffected in the normal eye. In the glaucomatous eye this mechanism for regulation is disturbed before other gross functional changes are present.

CAFFEINE TEST

In its simplest form this test is carried out by giving the patient a cup of strong coffee or by giving an intravenous injection of 0.2 Gm. of caffeine. Löhlein,²⁰ Wegner²¹ and others found that in glaucomatous eyes there is an increase of the intra-ocular tension that runs parallel to the increase in the blood pressure, while in the normal eye there is no variation (fig. 4).

20. Löhlein: *Klin. Monatsbl. f. Augenh.* **77**:909, 1926.

21. Wegner: *Zentralbl. f. d. ges. Ophth.* **24**:1, 1930.

VENOUS CONGESTION TEST

A tourniquet is placed around the neck just tight enough to cause venous congestion but without interfering with speaking or swallowing. The tension is taken before the procedure and at the end of an hour. An increase of more than from 5 to 6 mm. of mercury speaks for latent glaucoma. As a control, the tension is again taken ten minutes after the tourniquet has been removed. At this time, as a rule, the tension is below that taken before the start of the test. This test should not be applied to old patients or patients with advanced arteriosclerosis (fig. 4).

A simpler way of carrying out this test, according to Wegner,²¹ is to place the patient on a table with the head lowered from 20 to 30

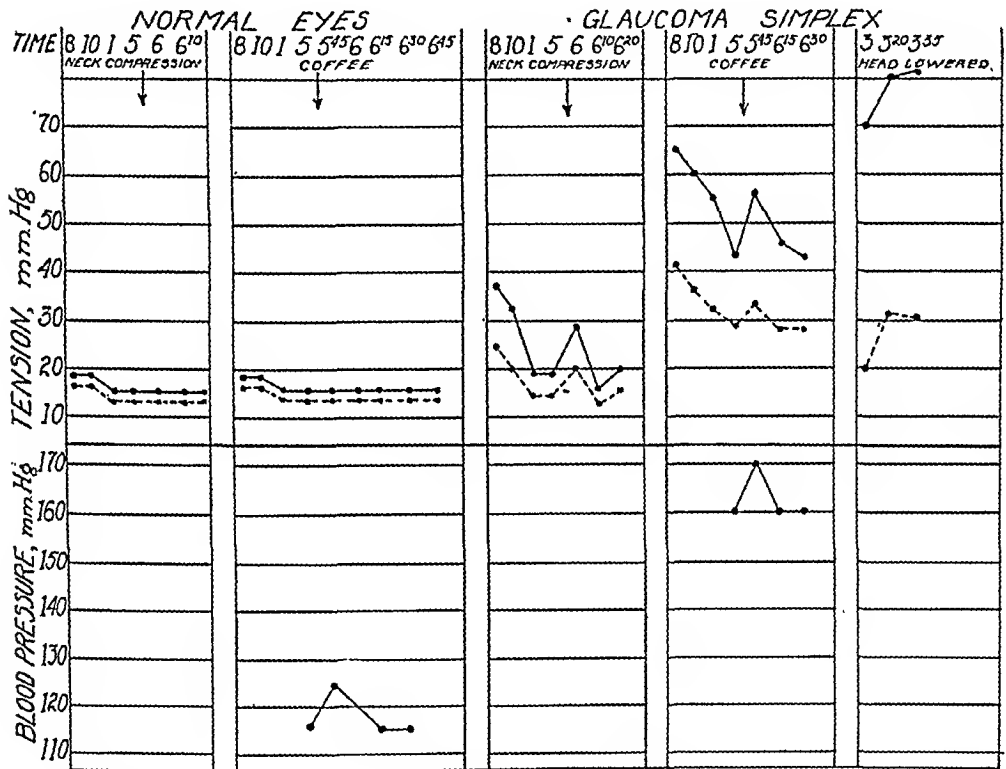


Fig. 4.—Curves for tension of normal eyes and glaucomatous eyes after compression of the neck, the drinking of coffee and lowering of the head.

degrees for from ten to fifteen minutes, when the tension, which has been taken before the test, is again taken. The variations already noted also apply with this method (fig. 4).

MASSAGE

A simple functional test is massage of the eyeball with the tonometer. If in the normal eye the Schiötz tonometer with the 5.5 Gm. weight is kept constantly resting on the eye for from three to five minutes, the tension will fall to one third or one half of normal and will return to normal in from thirty to forty-five minutes. In the glaucomatous eye

the tension may remain unchanged or may fall to a lower degree. The tension returns to its former reading much more rapidly than in the normal eye and may even exceed it and not return to its original reading for one hundred minutes (P. Knapp¹; fig. 5).

DARK ROOM TEST

According to Seidel²² and others, this test is very useful in latent glaucoma. The patient is put in the dark room for an hour after the tension has been taken. At the end of this time the tension is again taken. In glaucomatous eyes or in eyes with latent glaucoma there is a sharp rise. If the patient then looks at a brightly lighted white wall or at the clear sky the tension falls rapidly to its former level. The

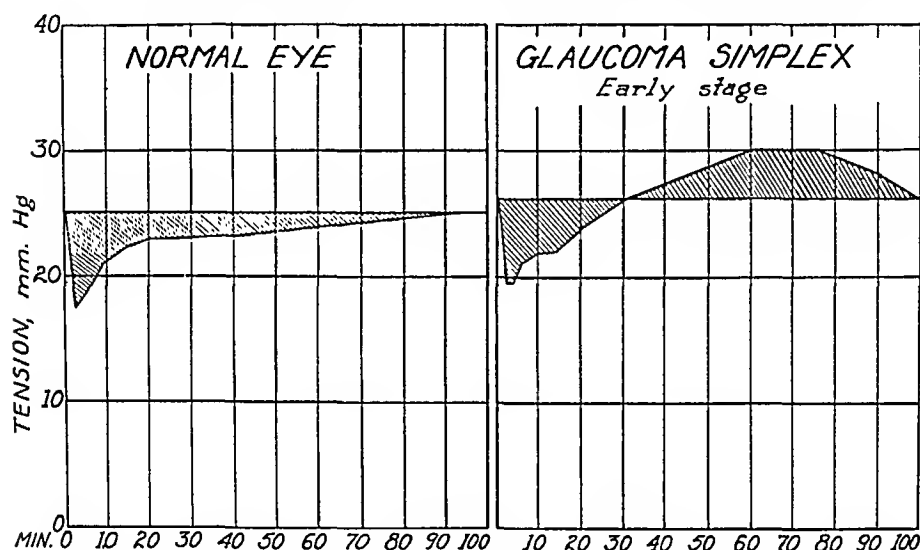


Fig. 5.—Curves for tension after massage for a normal eye and an eye in the early stage of glaucoma simplex.

variation may be from 10 to 40 mm. of mercury. In the normal eye this procedure causes no appreciable variation. This test is of particular value in the case of eyes the anterior chamber of which is shallow. The same procedure carried out in a dimly lighted room produces similar results (fig. 6).

The use of weak mydriatics will also cause the change in tension, but the sudden fall on exposure to light cannot be obtained.

YEARLY CURVE FOR SIMPLE GLAUCOMA

In simple glaucoma there is a variation in the yearly curve. If the tension is taken weekly, it is found that it is higher during the period

from November to January. The cause is not known. Perhaps the rapid change in temperature and atmospheric pressure play a part.

VISIBLE CHANGES IN THE EYE IN EARLY SIMPLE GLAUCOMA

In early simple glaucoma no visible changes may be present. In the beginning the anterior chamber is almost always normal. A fairly shallow chamber has doubtful diagnostic value, as the shallowness varies with the refraction and is greater in hyperopic and presbyopic eyes than in normal eyes. In glaucoma in persons under 35 (so-called juvenile glaucoma) the anterior chamber often is deeper than normal.

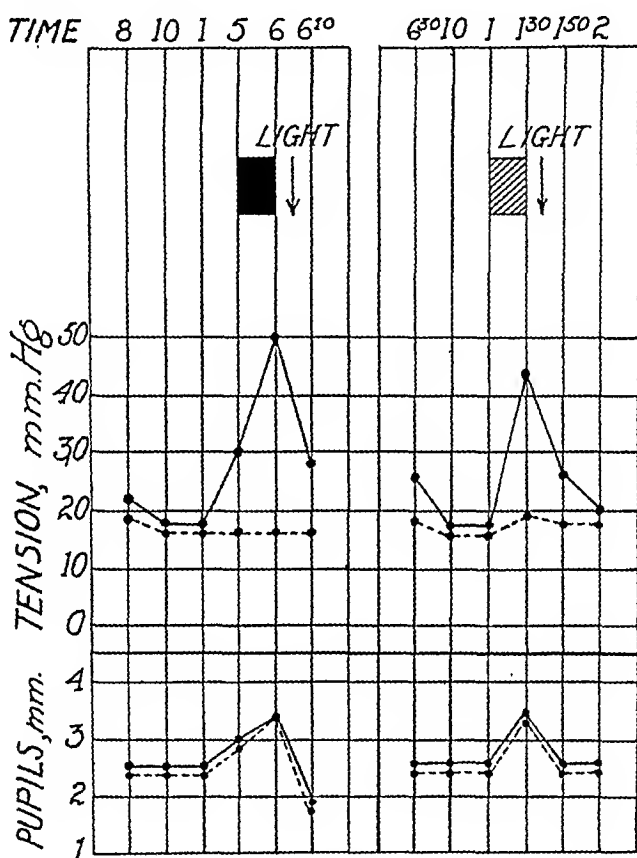


Fig. 6.—Curves for tension for a normal eye and a glaucomatous eye in the dark room test and in the test with dim light.

The size and reaction of the pupil are, as a rule, normal in the beginning. Pupillary changes, atrophy of the iris and increased congestion of the vessels of the iris make their appearance later in the disease. A. Knapp²³ reported that in many cases of early glaucoma instillation of 1 drop of a 1:1,000 solution of epinephrine hydrochloride caused pupillary dilatation, and he stated that this is an early symptom of compensated glaucoma.

23. Knapp, A.: Tr. Am. Ophth. Soc. 19:69. 1921.

With the slit lamp the presence of excessive fine pigment dust over the iris, the posterior corneal surface and surface of the lens is noted in glaucoma. Koeppe²⁴ expressed the belief that this is of great significance as indicating possible latent glaucoma. As these changes cannot be differentiated from the ordinary senile changes, their presence is of doubtful diagnostic value. Often they may appear later in the disease and especially after operative intervention.

Recently a good deal of work has been done on the angle of the anterior chamber in glaucoma. There is particular interest in the presence of pigment dust and its possible obstruction of Schlemm's canal. This pigment is often present in normal eyes to some degree. Probably the amount of pigment present has a bearing on this subject. O. Barkan²⁵ stated that on biomicroscopic examination he has seen in many cases of glaucoma an area of blockage in the trabecula which separates Schlemm's canal from the anterior chamber, and he feels this is a causative factor.

Opacities of the lens, as Gradle²⁶ has pointed out, occur so frequently during the age period when glaucoma is most prevalent that their presence is of no diagnostic value and must be regarded as entirely independent of the disease. Vogt²⁷ pointed out that in cases of lenticular opacity in which the opacity is primarily an exfoliation of the anterior capsule, glaucoma may develop. With the slit lamp the typical picture is seen, with bluish gray clumpings at the pupillary border of the iris. These are the result of exfoliation of the anterior capsule of the lens, and possibly these clumpings may also interfere with the drainage of Schlemm's canal and in this way cause glaucoma. This is so well established that the condition has been called glaucoma capsulare. In these cases there is usually low grade glaucoma that must be watched carefully.

The typical picture of glaucomatous evacuation and atrophy does not belong to the classification of early simple glaucoma but is seen only in the later states. The diagnosis of early glaucomatous cup is difficult. If there has been a definite physiologic cup in the center of the disk, this enlarges during the early stages of increased pressure. Consequently, in beginning glaucoma there may be only a large physiologic cup present in the disk. In every case of an unusually large physiologic cup, beginning glaucoma must be kept in mind. This condition should not be confused with a true glaucomatous cup, as the normal color is present, not the greenish tinge of the glaucomatous cup.

24. Koeppe, L.: *Ber. ü. d. Versamml. d. ophth. Gesellsch.* **40**:478, 1916.

25. Barkan, O.: Paper read before the American Academy of Ophthalmology and Otolaryngology, New York, Sept. 29, 1936.

26. Gradle, H., in Jackson, E.: *Contributions to Ophthalmic Science*, Menasha, Wis., George Banta Publishing Company, 1926, p. 255.

27. Vogt, A.: *Schweiz. med. Wchnschr.* **56**:413, 1926.

Also, the halo is absent, and there always remains at least a narrow band of normal nerve tissue. In cases in which a large physiologic cup is present careful sketches should be made so that one can determine from time to time whether it has increased in size. In cases in which no physiologic cup is present the earliest signs of excavation are a very flat shallow cupping beginning sharply from both the nasal and the temporal side. The vessels in this instance show only a very slight dip at the margin of the disk.

The time required to produce a glaucomatous cup varies greatly, depending on the degree of tension and the structure of the eye. There are cases in which the disk withstands rather high tension for a long time without signs of cupping, and, conversely, there are eyes in which the tension is relatively low and in which a glaucomatous cup develops early.

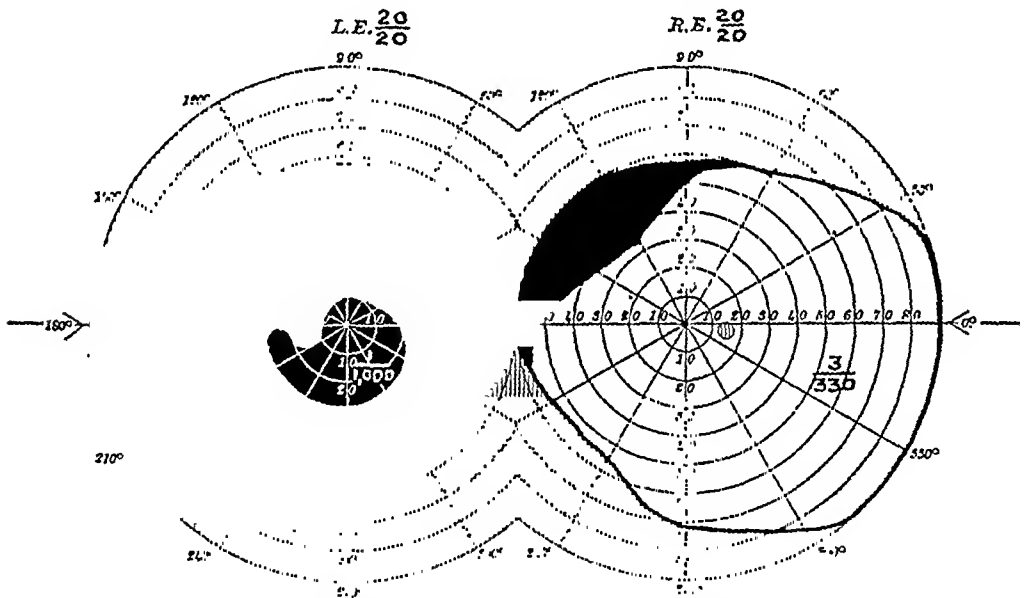


Fig. 7.—Visual fields at the beginning of primary simple glaucoma. The left eye shows an early cut central isopter as determined by the screen study; the right eye, an early cut nasal field, as determined by the perimeter.

CHANGES IN THE VISUAL FIELD

In primary simple glaucoma the disturbance of function is primarily the result of pressure on the optic nerve and its subsequent excavation. This atrophy of the nerve has the peculiar tendency to be involved by bundles, thus producing characteristic defects in the field.

At the beginning of the disease and even in the presence of a definite partial atrophy and excavation the peripheral field is usually normal except for a contraction of the nasal field, which may appear early in the disease. The contraction is not great and does not spread uniformly over the entire nasal field (fig. 7).

Very early in the disease scotomas extending from the blindspot are present, the form of which follows the course of the bundles of nerve fibers in the retina. Seidel²² found that in very early cases the so-called Seidel scotoma often was the only indication of the disease. This consists of a tuftlike or flamelike appendage to the blindspot or, when the condition is more advanced, of crescentic or sickle-shaped, narrow, pointed defects arching from one or both poles of the blindspot around the fixation area (fig. 8). These may be inconstant and vary from day to day or may develop into the typical Bjerrum scotoma. This consists of a semilunar scotoma extending from the blindspot above or below, or above and below (fig. 8). In the latter form the defects may meet in the nasal half of the field and produce a ring scotoma. The bundles of nerve fibers course about the fixation area in

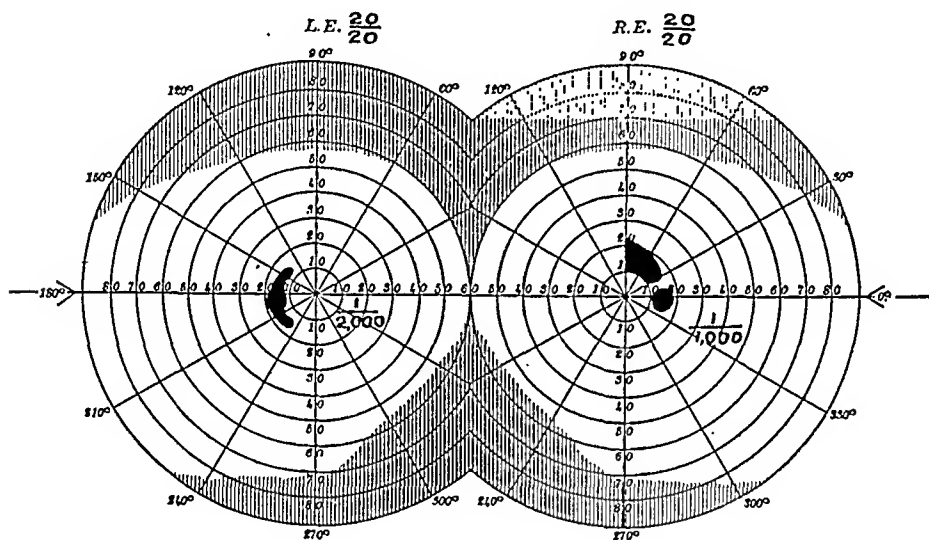


Fig. 8.—Visual fields in the early stage of primary simple glaucoma. The left field shows Seidel's scotoma; the right field, Bjerrum's scotoma.

the temporal half of the retina and end in the median raphe. As more damage is done to these fibers one observes a sharply defined defect in the nasal quadrant, the so-called nasal step of Rönne²⁸ (fig. 9). This is more frequently found in the lower quadrant or in both the lower and the upper quadrant, in the latter case producing a complete defect of the nasal field. This defect of the nasal field almost always arises from the center rather than from the periphery of the field. The contraction of the peripheral field usually occurs only in the later stages of the disease. At times the only change in the central field consists of definite enlargement of the blindspot without a defect in the peripheral field.

28. Rönne, H.: Arch. f. Ophth. 71:52, 1909.

From this it is apparent that the perimeter does not suffice in the study of early stages of glaucoma, and careful study on the screen is necessary in order to detect the earliest signs of glaucomatous changes in the field.

Central vision remains to the last, so that vision as determined by tests with Snellen's letters is no indication of the progress of the disease.

CHANGES OF REFRACTION

The most significant change in refraction in glaucoma is a rather rapid loss of accommodation, requiring frequent changes in reading glasses. Mild myopia may also occur, owing to forward displacement of the lens with decrease in the depth of the anterior chamber. A rapid change in presbyopia should always make one suspicious of glaucoma.

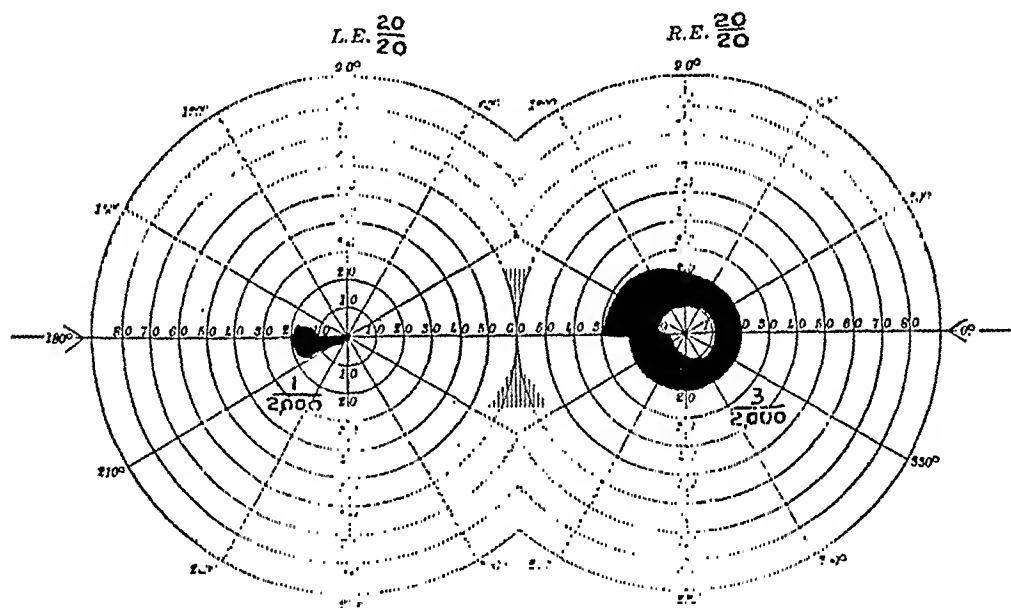


Fig. 9.—Visual fields showing later damage. The left field shows an atypical nerve fiber bundle defect; the right field, a double arcuate scotoma with Rönne's nasal step.

COURSE OF THE DISEASE

Chronic simple glaucoma begins without symptoms such as pain or visual disturbance, so that frequently the disease is well advanced when the ophthalmologist is consulted. If the disease is untreated the well known picture of glaucomatous cupping, atrophy and blindness ensues. There is also a tendency for glaucoma simplex to develop into the chronic inflammatory type of glaucoma. In unilateral glaucoma the apparently healthy eye must be watched carefully, as in the majority of cases both eyes are involved.

DIAGNOSIS OF SIMPLE GLAUCOMA

The presence of increased intra-ocular tension, excavation of the nerve head and atrophy and disturbances of function is sufficient to warrant a diagnosis of glaucoma. Until the aforementioned signs have been ruled out glaucoma cannot be excluded. The tension must be taken with a tonometer, as palpation is too unreliable.

In the early stages diagnosis is sometimes difficult. If increased pressure is present and there are scotomas of the type described, the diagnosis is certain, even though the nerve may show no visible change. The presence of normal tonometer readings in the middle of the afternoon does not exclude glaucoma in cases in which there is a scotoma associated with the blindspot, especially with an accompanying suspicious early change in the disk. Glaucoma cannot be ruled out until the twenty-four hour curve for tension has been recorded over a period of two or three days.

INCIDENCE

Primary glaucoma is a disease of old age, with a sharp rise in the curve for tension after 45 years of age, and is more frequent in women than in men. It is rather common among the Jewish race and is supposedly more frequent in the Orient than in Europe. It is also more frequent in the Negro race than in the white race. Its hereditary tendency has long been recognized. Both eyes are involved in most instances, and the condition has a tendency to run a similar course in the two eyes. It is often associated with arteriosclerosis and cardiovascular changes, which are probably coincident and are to be considered principally as additional signs of old age. The disease is more prevalent in hyperopes than in myopes.

MANAGEMENT OF SIMPLE GLAUCOMA

The treatment of simple glaucoma can be divided into two general headings: (1) medical and (2) surgical.

Medical Management.—The treatment discussed here is limited to that for early simple glaucoma.

In the medical care of glaucoma both the oculist and the patient assume a great responsibility. It is necessary to impress on the patient the seriousness of the disease and the necessity of constant medication and periodic observation. In the periodic examination, tension, examination of the fundus and studies of the field are important. Of these, the most important is the careful detailed screen study of the defect of the field. The changes in the field are the most important indication as to the progress of the disease, as some eyes can tolerate a tension slightly higher than normal without change while in some an apparently normal tension is sufficient to produce changes.

Miotics—Pilocarpine and Physostigmine: In spite of recently advocated medication, pilocarpine and physostigmine still hold the first place in importance. Their action is to stimulate the parasympathetic end-organs of the oculomotor nerve, producing contraction of the pupil. The most commonly accepted theory of their action is that the base of the iris is freed and the angle of the anterior chamber made more open, thus opening Schlemm's canal, and this increases the absorbing surface of the iris.

Pilocarpine hydrochloride or pilocarpine nitrate is given in a 0.5 to a 2 per cent solution, depending on the action desired. In some patients who are sensitive a 0.5 per cent solution will keep the pupil contracted for from eight to twelve hours. With the average patient a 1 or a 2 per cent solution of pilocarpine given every four to six hours controls the tension during the day. It is advisable to take the tension at the end of the four to six hour period to determine whether or not it has risen above the normal, in other words, to determine whether the drops maintain the normal tension for that period of time. On the basis of the knowledge that the tension is apt to rise during the night, a drop must be instilled just before retiring. In patients for whom the curve for the tension shows a definite rise at night, an ointment containing from 1 to 2 per cent of pilocarpine applied at night just before retiring is indicated. In the group in whom the tension is normal during the day but rises at night, a 1 to 2 per cent solution of pilocarpine instilled at night just before retiring is often sufficient. In some instances a pilocarpine ointment at night controls the curve for tension efficiently. In these cases, particularly, careful periodic examination of the central fields is important.

Physostigmine salicylate or physostigmine sulfate can be employed in a 0.2 to a 0.5 per cent solution. The action of physostigmine is stronger than that of pilocarpine, but it has the disadvantage of being slightly irritating and in most patients causes a dermatitis venenata in a relatively short time. It is pretty generally accepted that long-continued physostigmine therapy is detrimental to the eye, so that any case in which the use of physostigmine is required for the maintenance of normal tension must be considered a surgical one.

Pilocarpine also, after being used over a long period, is prone to produce a dermatitis with accompanying conjunctivitis. When this occurs the patient is treated with solution of physostigmine for three or four days, during which time the dermatitis and conjunctivitis disappear. It is a good rule to have the patient use solution of physostigmine as a routine for two or three days of every month.

In cases in which miotics do not control the tension, surgical intervention, as a rule, is indicated. In such cases the sooner surgical

intervention is advised the better. Certain circumstances, however, may make a delay advisable or operation impossible, and under these conditions some of the more recently advocated therapeutic measures may be indicated. Some of these conditions are: a very old and feeble patient, refusal of operation, a one-eyed person who refuses operation.

Epinephrine: Of the newer therapeutic agents, epinephrine and its derivatives are most useful. Wessely²⁹ was the first to show experimentally the effect of epinephrine on the intra-ocular tension. He found that both simple instillation and subconjunctival injection of epinephrine hydrochloride reduced the tension in the eyes of animals. Ordinary epinephrine hydrochloride instilled is effective in only a small percentage of cases, but subconjunctival injection is followed by a fall in the tension in most cases that reaches its maximum from twelve to fourteen hours after the injection, the dose being 4 minims (0.24 cc.) of a 1:1,000 solution of epinephrine hydrochloride injected under the conjunctiva at the temporal side of the globe. This causes a rise in blood pressure, which in some patients is accompanied by systemic symptoms.

I have found the method of Gradle³⁰ effective a good many times. This produces almost as much effect as an injection but without systemic reactions. The conjunctival sac is anesthetized, and from 4 to 5 minims (0.24 to 0.3 cc.) of a 1:1,000 solution of epinephrine hydrochloride is dropped on a small cotton pledget, placed in the upper cul-de-sac and left for two and one-half minutes.

Hamburger³¹ advised synthetic dextrorotatory epinephrine, which he called *Glaukosan*, for the injection. To avoid injection he used a 2 per cent solution of epinephrine hydrochloride, to which he added an optically inactive substance produced in the manufacture of epinephrine, which he called *Links-Glaukosan* or *Laevo-Glaukosan*. Two drops of the solution are placed in the inner angle, and the lids are held open for thirty seconds. This is repeated from three to five times. More recently suprarenin bitartrate in a 2 per cent solution has been used in this country, and within the past few months I have had occasion to use a new 1:100 solution of epinephrine hydrochloride, which is more stable than any of the synthetic epinephrine products.

All these methods apparently have practically the same effect on the tension. In most cases they produce a fall of the tension below 25 mm. (Schiötz). It is necessary to use miotics after the treatment in the usual manner. The use of epinephrine in conjunction with miotics may keep the tension normal for from a week to as long as six weeks. Often

29. Wessely, K.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **28**:69, 1901.

30. Gradle, H.: The Use of Epinephrine in Ocular Hypertension, J. A. M. A. **84**:675 (Feb. 28) 1925.

31. Hamburger, C.: Klin. Monatsbl. f. Augenh. **76**:400, 1926.

later treatments are not as effective as the first treatment, and there is a marked variation in response to the treatment. If the first treatment is ineffective, further attempts are useless.

The possibility of acute glaucoma resulting from the use of these epinephrine preparations must be kept in mind, even though this is infrequent in simple glaucoma. The use of physostigmine before the treatment and every twenty minutes for the first hour after the administration of epinephrine seems to help prevent this. In my experience the use of epinephrine wicks seems less likely to produce this complication. According to Gifford,³² the possibility of an acute attack speaks against the use of epinephrine ointment at home.

Other Drugs.—Ergotamine tartrate (gynergen), a derivative of ergot, supposedly depresses the end-organs of the sympathetic nervous system and thus should help glaucoma by decreasing the permeability of the vessels. The dose is one-half ampule for injection or $\frac{1}{60}$ grain (0.001 Gm.) by mouth. Injections are given twice daily, or the oral dose is given two or three times a day. This treatment appears to help in some cases but is very expensive and, in my opinion, does not offer sufficient advantage over other methods to warrant its use.

Recently extract of adrenal cortex has received a good deal of publicity. My experience with its use in a relatively few cases has been that it has no effect on the intra-ocular tension in glaucomatous eyes. There have been several reports in the literature confirming this experience. Woods³³ found adrenal cortex extract ineffective in glaucoma, whether given intramuscularly or intravenously.

The effect of any of the aforementioned therapeutic agents, aside from miotics, is a temporary one, and their value in simple glaucoma is chiefly in cases in which operation must be delayed.

GENERAL CARE

The patient should have a careful physical examination, and measures should be taken to control the blood pressure and obvious pathologic conditions. The so-called vasoneurotic diathesis (unstable vasomotor mechanism, i. e., tachycardia, angioneurotic edema and urticaria) and the related bronchial asthma seem to have a relationship to glaucoma. All frank foci of infection should be cared for. One is not justified, however, in recommending radical procedures, such as the removal of tonsils suspected to be infected. The patient should also be warned against physical and mental excitation. The average use of the eyes for near work can also be allowed.

32. Gifford, S. F.: *Am. J. Ophth.* **11**:628, 1928.

33. Woods, A. C.: *The Use of an Extract of Adrenal Cortex in Glaucoma*, *Arch. Ophth.* **14**:936 (Dec.) 1935.

Surgical Management.—In cases in which miotics do not control the tension, surgical intervention is indicated. The most important indication of the progress of the disease is changes in the field as determined by a careful, detailed screen study. Any increase in the size of the scotomas is an indication for operation when the tension has not fallen to well within the normal limits.

Cyclodialysis is the operation of choice for early simple glaucoma when the tension is not above 40 mm. (Schiötz). If properly done it opens up a space between the sclera and the choroid for the absorption of aqueous. The severing of the ciliary nerves and vessels over the area of the operation also reduces the formation of the aqueous. I have found the operation successful in a high percentage of properly selected cases. Meller³⁴ stated that he obtained successful results in about 70 per cent of his cases. In cases in which the procedure has not been successful it can be repeated two or three times in different quadrants. In performing the operation certain details have aided in increasing the percentage of successful results. The patient is given a preparation of calcium phosphate and calcium lactate for three days preoperatively to reduce the bleeding to a minimum, the dose being a teaspoonful in a glass of water three times a day before meals. The scleral incision is made at a measured distance of from 4 to 4.5 mm. from the limbus. In making the incision the keratome is tilted away from the limbus; this makes possible an oblique incision through which the spatula is easily introduced into the anterior chamber. The tip of the spatula should be introduced almost to the pupillary area so that separation is obtained over a large area. The operation is not difficult, and should it fail it in no way interferes with any of the filtering operations. In addition, it does not have the disadvantage of the filtering operations, such as the production of sudden drops of tension, with resultant retinal hemorrhage or the possibility of late infection. On rare occasions there is hypotony with associated lenticular changes. These conditions, however, occur in so few cases that they can be practically disregarded. When the operation is successful the intra-ocular tension is reduced to normal; miotics, however, must be continued to assure continued control of the tension.

In cases in which cyclodialysis is unsuccessful one of the filtering operations must be resorted to, the type of the operation depending primarily on the choice of the operator.

It is possible that in the future the goniotomy operation of O. Barkan may prove to be the proper procedure in glaucoma simplex.

34. Meller, J.: Personal communication to the author, 1935.

PROGNOSIS

In early glaucoma simplex the prognosis is rather good, and in many patients vision can be preserved throughout life by miotics controlled by careful periodic observation. Even in the early stages of the non-malignant type for which surgical intervention is required, the outlook for retention of vision is good. In all cases, however, continued periodic observation is necessary. The most important indication of the progress of the disease is the changes in the field of vision, particularly changes in the central field, as noted in studies on the screen.

Obituary

WARD ANDREWS HOLDEN, M.D.

1866-1937

Ward Andrews Holden died suddenly on January 24, at the University Club, New York, in his seventy-third year. His death removes another outstanding figure from American ophthalmology.

He was born in Marietta, Ohio, the son of William and Sarah Andrews Holden, on Feb. 15, 1866. He received the degree of Bachelor of Arts in 1884 from Marietta College, of which his grandfather, Israel Ward Andrews, was president; and the degree of Doctor of Medicine from the Medical College of Ohio, Cincinnati, in 1887. After taking postgraduate work at the University of Göttingen, the University of Vienna and the University of Marburg, from 1888 to 1890, he settled in New York and became associated with Dr. Herman Knapp in his private office. He was connected with the New York Ophthalmic and Aural Institute, and the Vanderbilt Clinic of the College of Physicians and Surgeons. From the first Holden gave much attention to work in the laboratory, where he gave courses for a number of years on the embryology and pathology of the eye; he wrote short textbooks on each of these subjects in 1893. At the Pathological Institute of the New York State Hospital he made his important investigations on toxic amblyopias, in which he demonstrated the degeneration of the retinal ganglion cells, findings which were later verified by Birch-Hirschfeld. This work was published under the following headings: "Experimental Quinine Amblyopia" and "Pathology of Amblyopia Following Profuse Hemorrhage and of That Following the Ingestion of Methyl Alcohol, with Remarks on the Pathogenesis of Optic Nerve Atrophy in General."¹ In 1898 and 1899 he published two articles, "A Case of Excessive Distortion of the Optic Chiasm in Acromegalia" and "The Sequence of Changes in the Optic Chiasm Produced by Acromegalia as Exemplified in Three Cases."² These were among the earliest publications on the important relation of the eye to the pituitary region.

1. Holden, W. A.: Experimental Quinine Amblyopia, *Arch. Ophth.* **27**:457, 1899; Pathology of Amblyopia Following Profuse Hemorrhage and of That Following the Ingestion of Methyl Alcohol, with Remarks on the Pathogenesis of Optic-Nerve Atrophy in General, *ibid.* **28**:125, 1899.

2. Holden, W. A.: A Case of Excessive Distortion of the Optic Chiasm in Acromegalia, *Arch. Neurol. & Psychopath.* **1**:699, 1898; The Sequence of Changes in the Optic Chiasm Produced by Acromegalia, as Exemplified in Three Cases, *ibid.* **2**:575, 1899.

He then became interested in the association of the eyes with nervous diseases, and served as attending ophthalmologist to the Neurological Institute from its beginning on Sixty-Seventh Street, where he worked faithfully until the institute moved up to its new quarters.

In 1904 Holden became chief of clinic in the ophthalmic department of the Vanderbilt Clinic and honorary surgeon to the Herman Knapp Memorial Eye Hospital.



WARD ANDREWS HOLDEN, M.D.

1866-1937

In 1918 he wrote a short paper, "The Eye in Nervous Disease: An Introduction to Neurological Ophthalmology, prepared for the New York Neurological School for Army Medical Officers."³

3. Holden, W. A.: The Eye in Nervous Disease: An Introduction to Neurological Ophthalmology, Prepared for the New York Neurological School for Army Medical Officers, Arch. Ophth. 47:221, 1918.

As one of the editors of the *Archives of Ophthalmology* from 1893 to 1928, he did yeoman service in the cause of scientific ophthalmology in this country.

Holden was of a quiet and retiring nature. He had marked ability and many interests of an artistic nature outside of his profession which made an impress on his medical thought and action. He had an unusually alert mind, and his literary style was lucid. He never took an active part in medical meetings but succeeded in making real contributions to ophthalmology, for which his name will always be remembered. He was fond of music and art and had marked ability in drawing. This talent proved of great help in his profession and later furnished activity during many summer holidays, which usually resulted in a series of delightful pastel sketches.

Holden was one of the original members of the Charaka Club, a club devoted to the historical, literary and artistic sides of medicine. He never married, and for many years the University Club was his second home; it seemed fitting that he should have been listening there to a symphony concert on the radio when the fatal stroke of paralysis came. He left no near relative except a sister, Mrs. Elizabeth Holden Canda, of Pomfret, Conn.

ARNOLD KNAPP.

* * * * *

When I returned from Germany in the early nineties, whither I had gone to study neurology, I realized that in the diagnosis of nervous diseases the use of the ophthalmoscope was essential. Hence I offered myself as voluntary assistant in the New York Ophthalmic and Aural Institute, which was under the direction of the leading ophthalmologist of the country, Dr. Hermann Knapp. I soon found there a young man who was earnest, efficient and affable. I stuck to him as a burr does to the bushy tail of a dog. From those days dated a friendship that existed nearly half a century. I had not been in the outpatient department of the hospital long before he began to give me instruction. After the work of the afternoon was finished we would walk uptown. The hospital was at Twelfth Street, and we both lived in Thirty-Eighth Street. On the way we were likely to stop at one of the book shops which were then in Union Square. I soon found that he knew not only his mother tongue but German, Italian and French, and as I, even in those early days, was interested in letters, we had many interesting discussions. I use the word discussion, but imparting of information would be a more correct term. He made no parade of his knowledge of literature, but he was ready to reveal it on request. Many of our evenings in the winter we spent in the gallery of the Metropolitan Opera House or listened to the New York Philharmonic Symphony or other symphony societies. He had the same sensitiveness for music

that he had for literature and art, and by art I mean painting. All that I know of pictures he taught me. Even in our later life he would take me from gallery to exhibition and point out the merits or the demerits of the exhibits. He was himself a water colorist of high order, and it was a pleasure to observe him showing his pictures so modestly to the members of the Charaka Club. When Joseph Frankel and I established the Neurological Institute, Dr. Holden became the attending ophthalmologist, and he worked hard, faithfully and efficiently to our great advantage for nearly fifteen years.

Dr. Holden was what the French call a savant, that is, a person who knows all there is to be known about the subject of his profession and in addition knows something about nearly everything else. His outstanding characteristics were courtesy, decorum, unselfishness and modesty. Therefore, he was a gentleman; in fact, of all the gentlemen I have known, Ward A. Holden tops the list.

JOSEPH COLLINS.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

RETINAL METABOLISM. R. CAMPOS, *Ann. di ottal. e clin. ocul.* 64: 457 (July); 538 (Aug.); 577 (Sept.) 1936.

The work reported, which received the Cirincione prize for 1935, included studies of the respiration of the retina in the rat, the rabbit and a few human beings. The Warburg apparatus was employed for both the direct and the indirect methods. The literature on previous work on the retina by Warburg and others is reviewed.

In the work with the rat and the rabbit a marked difference was found between the respiration estimated by the direct method and that estimated by the indirect method. The high figures obtained by the indirect method represent something peculiar to the retina, as compared with other tissues, which is apparently the result of glycolysis. When the retina was first dark-adapted, the consumption of oxygen was much increased, as compared with that in the light-adapted retina. These findings were confirmed in the monkey, and Campos considers this as proof of Ovio's theory that adaptation is an expression of the retinal metabolism.

The gas exchange of the human retina was examined by both the direct and the indirect method. The material was eyes removed from six to ten days after a penetrating wound, one normal eye and three eyes with secondary glaucoma. There was little change shown in the results following wounds for periods up to ten days. The retina in all these eyes showed an active respiration, with marked aerobic and anaerobic glycolysis, the former being more marked than the latter. There was no excess of fermentation, and the Pasteur reaction was low or negative. The findings agreed with those found in normal rabbits' eyes. In eyes with further changes in the retina such as occurred from twenty-four hours to ten days after injury, aerobic glycolysis was reduced, while anaerobic glycolysis remained nearly normal.

The macular region was compared with the peripheral part of the retina in its respiration. The peripheral part of the retina showed a more active respiration in the relatively normal eye, while in an eye with absolute glaucoma this condition was reversed.

The choroid and retina of pigmented and albino rats were investigated by the Warburg method. The choroid of albino animals showed the more active respiration. The retina of the albino rats showed a slightly more active respiration, while its aerobic glycolysis was definitely less than that of pigmented animals. A bibliography is included.

S. R. GIFFORD.

Blind

BLINDNESS IN WEST CHINA. E. R. CUNNINGHAM, Chinese M. J. 50: 1507 (Oct.) 1936.

In this paper brief mention is made of reports and statements relating to blindness, published by ophthalmologists working in China. A list is given of the causes of blindness found in 749 blind eyes in 596 patients. It is pointed out that the acute ophthalmias which are so closely associated with corneal ulceration are undoubtedly at the bottom of most of the blindness. Trachoma also plays a large part and is often allied with acute conjunctivitis. When economic conditions are disturbed, keratomalacia may produce much blindness.

Blindness caused by congenital or hereditary disorders was met with only rarely.

S. H. McKEE.

Conjunctiva

A RETENTION CYST OF UNUSUAL SIZE, PROBABLY OF KRAUSE'S GLAND. SIMULATING ANGIOMA OF THE ORBIT. D. V. GIRI, Brit. J. Ophth. 20: 621 (Nov.) 1936.

A cystic tumor which was smooth, mobile and nonadhesive to the skin, situated at the upper inner angle of the orbit, was seen in a girl of 9 years. On removal it proved to be a white translucent oval cyst about 3 cm. long and 1.7 cm. broad. Histologically the cyst was lined with two layers of cells, a deeper layer of flattened and a more superficial layer of cubical cells. In parts there were several layers of cells resembling those of altered conjunctiva. There was a well marked wall of fibrous tissue, and here and there were nodules of lymphocytes. The diagnosis was a probable retention cyst arising from the conjunctiva or a gland of Krause. The growth was not an ordinary dermoid.

The article is illustrated.

W. ZENTMAYER.

HISTOLOGIC EXAMINATION OF PROWAZEK'S BODIES AND STATISTICS ON THEIR GEOGRAPHIC DISTRIBUTION. Y. ONISI, Klin. Monatsbl. f. Augenh. 96: 797 (June) 1936.

In the first chapter of this paper the histologic technic of the preparation of Prowazek's bodies is given, and their relation to trachoma and to inclusion blennorrhoea is discussed. The second chapter contains elaborate statistical data and tables on the geographic distribution of Prowazek's bodies in the northern part of Japan. Smears and sections in 1,448 cases of trachoma were examined for a period of five years. Prowazek's bodies were found in 54 cases, or 3.73 per cent. This percentage was lower than in the southern districts of Japan. Onisi therefore concludes that the southern climate is more favorable for the development of Prowazek's bodies. In most of the cases in which these bodies were present severe papillary hyperplasia of the conjunctiva was found clinically.

K. L. STOLL.

Cornea and Sclera

THE PATHOLOGY OF LATTICE AND NODULAR DYSTROPHY OF THE CORNEA. F. H. MAURY, *Am. J. Ophth.* 19: 866 (Oct.) 1936.

Maury reports a case of bilateral corneal dystrophy in a 58 year old man one of whose eyes was phthisical, apparently as a result of surgical intervention. As this eye did not have useful vision and was persistently painful, it was enucleated and examined. Maury gives the histologic findings and reviews the literature from the standpoint of the pathologic reports. He gives the following summary:

"The clinical and histological findings in a case of combined lattice and nodular dystrophy of the cornea are reported. The lattice and nodular lesions both showed the same histological features. The primary lesion is a hyalin-lipoid degeneration of localized portions of the corneal lamellae."

W. S. REESE.

GUMMA OF THE CORNEA. A. VOLKOVA, *Sovet. vestnik oftal.* 9: 350, 1936.

The cases of two young female patients with gumma of the cornea are presented because of the rarity of the condition. The following were the outstanding features: parenchymatous keratitis and a yellow-gray tumor with sharply demarcated borders at the site of the densest opacity of the cornea. The tumor gradually became vascularized and elevated 1 mm. above the surface of the cornea. In the first patient it had the shape of a mushroom cap; in the second patient the shape was oval. In the first patient the Wassermann reaction of the blood was negative at first and became positive after a provocative injection of neoarsphenamine. The gumma absorbed after six injections of neoarsphenamine. In the second patient the gumma underwent partial necrosis associated with marked hypotony, because of the fistulization of the cornea. In both patients a milky-white scar formed at the site of the gumma after a period of several months of antisyphilitic treatment.

O. SITCHEVSKA.

CONTRIBUTION TO THE KNOWLEDGE OF THE MARGINAL CORNEAL ULCER. P. S. SOUDAKOFF, *Chinese M. J.* 50: 1393 (Oct.) 1936.

Five cases of ring-shaped marginal corneal ulcer are described. In one case the condition was diagnosed as an endogenous infection and in the other four cases as an exogenous infection. In three cases of the latter group pathogenic micro-organisms were isolated, while in one case the causative micro-organism was not established.

The ring-shaped marginal ulcer usually has a moderately severe course, with tendency to recurrence. In exceptional cases, as in the four reported in this article, the ulcer was progressive and the outcome was a complete leukoma of the cornea.

The clinical facts indicate that the cornea that has once been affected by a ring-shaped ulcer is predisposed to recurrence at the inner border of the previously formed scar. There is a considerable bibliography.

S. H. MCKEE.

Experimental Pathology

PRECIPITINS IN THE OCULAR TISSUES OF RABBITS GENERALLY AND LOCALLY IMMUNIZED WITH CRYSTALLINE EGG ALBUMIN. R. THOMPSON, E. GALLARDO and D. KHORAZO, *Am. J. Ophth.* 19: 852 (Oct.) 1936.

This article does not lend itself to abstracting. Five tables are given showing the results of experiments on albino rabbits. The animals were immunized intravenously or given single or repeated local injections of crystallized egg albumin into the anterior chamber or into the cornea. The authors remarked the presence, following the local injection of antigen, of precipitins in the corneal tissue earlier and in greater concentration than elsewhere. Their finding of the absence of precipitins in the aqueous until the serum contained them and until local inflammation had occurred corroborates the results noted in previous work. These experiments indicate that the aqueous does not reflect the state of the surrounding tissues.

W. S. REESE.

OCULO CARDIAC REFLEX AS MODIFIED BY INDUCED HYPERTHERMIA. P. VALLERY-RADOT, G. MAURIC and J. LEMANT, *Compt. rend. Soc. de biol.* 123: 670, 1936.

In a series of subjects hyperthermia was induced by intramuscular injections of a 1 per cent solution of sulfur in oil, and the cardiac reaction to ocular compression was studied. Under average normal conditions the pulse rate is reduced four to twelve beats, but in the cases of hyperthermia the oculocardiac reflex became attenuated, obliterated or reversed. The authors conclude that hyperthermia provokes a tendency to sympathicotonia, which may persist for from three to six days after the curve for temperature has become level.

J. E. LEBENSOHN.

General Diseases

PERNICIOUS ANEMIA WITH RETROBULBAR NEURITIS. G. TALBOT, *Brit. J. Ophth.* 20: 619 (Nov.) 1936.

A man aged 55 years had pernicious anemia, which had been recognized and treated over a period of seven years. Vision had been failing for one year in each eye and equaled counting fingers. The optic nerve did not show atrophy, but one disk was a little paler than the other. The visual fields were full but showed a central relative scotoma. The pupils reacted to light.

Talbot suggests that the ocular condition might be accounted for by a demyelination of the optic nerve comparable to the demyelination of the posterolateral columns found in subacute combined degeneration. The comparatively slight interference with the axis-cylinders would account for the nondevelopment of optic atrophy.

W. ZENTMAYER.

SECONDARY ZONULAR OPACITY OF THE CORNEA IN POLYARTHRITIS CHRONICA LEUKOCYTOTICA (STILL) AND LYMPHOCYTOTICA (RHONHEIMER). J. KARSCH, *Arch. f. Augenh.* 110: 106, 1936.

Polyarthritis chronica leukocytotica, or Still's disease, is an infectious disease of childhood of unknown etiology. The signs are fever,

enlargement of the spleen and the lymph glands and an inflammation of the small joints in the form of peri-arthritis. The bones and cartilages often remain normal for years. The fluid in the cavities of the joints contains polymorphonuclear leukocytes. The disease usually begins at about the age of 8 and may last for years. Adhesive pericarditis frequently develops.

The form described by Rhonheimer differs but little from this form, with the exception that the fluid aspirated from the joints contains lymphocytes.

Karsch reports two cases of this disease in both of which there was an old iridocyclitis and zonular opacity of the cornea. Syphilis and tuberculosis could be excluded from the diagnosis. He considers the involvement of the cornea and iris as part of the picture of the disease.

F. H. ADLER.

Glaucoma

CYCLODIALYSIS IN THE TREATMENT OF CHRONIC GLAUCOMA. BAILLIART and LAIGNIER, Bull. Soc. d'opht. de Paris, October 1935, p. 636.

Cyclodialysis, which is used in Poland, Germany, Austria, Hungary and Czechoslovakia in the treatment of chronic glaucoma, is rarely the technic in France, because of the influence of Lagrange and his method of sclerotomy. From their use of cyclodialysis and from the discussion of the procedure as found in the literature, Bailliart and Laignier conclude that it is a simple operation, inoffensive and, without doubt, efficacious. Its action is not as lasting as sclerotomy, but it may be repeated, with little danger.

L. L. MAYER.

NEWER KNOWLEDGE OF THE DIAGNOSIS AND THERAPY OF PRIMARY GLAUCOMA. A. BRÜCKNER, Schweiz. med. Wchnschr. 66: 1264 (Dec. 12) 1936.

Brückner emphasizes the hereditary nature of glaucoma, showing how it may attack successive generations in the same decade of life. The theory of edema with changes in the p_H of the intra-ocular fluids as observed by Fisher and by Meesmann is considered. The influence of the vegetative nervous system and of climatic conditions is alluded to. Early subjective symptoms of the insidious chronic glaucoma may be a feeling of fulness in the eye or headache. Various tests to be made in the preglaucomatous stage are outlined, and the importance of the taking of tension periodically during the twenty-four hours of the day is emphasized. In the treatment the usual miotics at times control tension. A choline derivative which affects the parasympathetic nervous system has been of great success in Brückner's hands. Various operative procedures are discussed, and the indications for each type are outlined. The injection of alcohol relieves the pain in cases of absolute glaucoma.

L. L. MAYER.

Hygiene, Sociology, Education and History

A NEW EYE SHIELD FOR USE IN INDUSTRY. C. G. KAY SHARP, Brit. J. Ophth. 20: 683 (Dec.) 1936.

The factors which a miner's eye shield must embody are: no diminution of illumination; free ventilation in front of the face, in use or out of use, without removal; strength; light weight, and ease with which the apparatus can be put on and taken off. Sharp has devised a shield embodying these factors. It consists of a fiber, flexible head-band with a sliding adjustment to fit any head. On the front of the head-band is a metal plate to which is fitted a metal projection carrying a double ball and socket joint. At the distal end of this joint is an oblong metal frame carrying laminated glass, in the case of the miner's protector, and mica or gauze in other trades.

W. ZENTMAYER.

Injuries

NOTES ON AN EYELASH CARRIED BY A PERFORATING INJURY INTO THE POSTERIOR AQUEOUS CHAMBER AND REMOVED ELEVEN WEEKS LATER. L. H. SAVIN, Brit. J. Ophth. 20: 609 (Nov.) 1936.

Following a perforating wound in the upper nasal quadrant of the cornea the iris became attached to the whole posterior length of the wound. About seven weeks later an attempt was made to divide the anterior synechiae with de Wecker's scissors. Later it was seen that a fine strand remained. With a Ziegler knife needle an attempt was made to divide the strand, but as it was not taut it was pulled on by the knife, and an eyelash was drawn down from behind the iris. The lash was subsequently removed through a keratome incision. The eye recovered, with vision of 6/9.

W. ZENTMAYER.

INDUSTRIAL EYE INJURIES AND THEIR PREVENTION. J. MINTON, Brit. J. Ophth. 20: 673 (Dec.) 1936.

This paper is based on a study of patients who were seen in the casualty department of the Royal Eye Hospital, London. In 1935, 10,786 patients were seen in the casualty department, of whom approximately 6,500 were suffering from industrial injuries of the eye. Foreign bodies in the cornea formed the bulk of these injuries. Approximately 95 per cent of these patients were occupied in metal and engineering trades. Foreign bodies in the subtarsal region were rare and were seen only in those occupied in loading and transport trades. Corneal abrasions formed the next largest group of injuries. Twenty-seven patients had intra-ocular foreign bodies. Four eyes had to be excised; traumatic cataract developed in 13. Ten patients were discharged with vision of 6/12 or better. Twenty-two cases of perforating injury to the eye were observed. Seven eyes had to be excised; 15 patients were discharged with a vision of 6/18 or better. Chemical burns were comparatively rare, probably because there are few chemical factories in the vicinity of the hospital. Injuries from lime, concrete and cement were about as common as perforating injuries. Conjunctivitis in persons engaged in electric welding was uncommon. In nearly all

the cases of this condition the patients either had neglected to use the goggles provided or had not kept their heads behind the protective screen.

Minton discusses the extent of industrial injuries of the eye and their prevention.

W. ZENTMAYER.

VASCULAR CHANGES IN THE NORMAL FUNDUS PRODUCED BY SHORT WAVE THERAPY. E. TROVATI, *Ann. di ottal. e clin. ocul.* 64: 603 (Sept.) 1936.

Patients were treated with two forms of apparatus, one delivering a wavelength of 30 meters and the other, a wavelength of 8 meters. Electrodes measuring 8 by 14 cm. were applied to the sides of the head for from twenty to thirty minutes. Observations were made before, during and after treatment. Ten patients with normal fundi were treated. All the patients showed evidence of vasodilatation, which was about the same with the two types of apparatus. This was seen either as hyperemia of the disk or as definite enlargement of both the veins and the arteries on the disk. It was seen after ten minutes of treatment and persisted until five hours afterward. No signs of hyperemia were visible after twenty-four hours. These signs of hyperemia suggest that the use of short-wave therapy would be dangerous in glaucoma but of possible value in certain ocular diseases due to abnormal vascular conditions. In such cases the results, which will be reported later, were encouraging.

S. R. GIFFORD.

Lacrimal Apparatus

PATHOLOGIC HISTOLOGY OF THE LACRIMAL GLAND. H. HERKEN, *Arch. f. Augenh.* 110: 61, 1936.

The lacrimal glands were obtained at autopsy from one hundred and eight patients and examined histologically. Large amounts of fat were observed in the epithelial cells in the majority of cases; this does not represent a pathologic process. In senile glands pigment was frequently noted, together with the fat. In cases of tuberculosis, generalized infections, carcinoma and chronic diseases excessive amounts of fat were present.

In sixty patients there was definite lipomatosis, usually combined with atrophy of the cells of the gland. Forty of these patients were over 50 years of age. Atrophy of the gland can occur with or without replacement by connective tissue. All the patients over 50 years of age showed hyperplasia of connective tissue.

The lacrimal gland of the new-born does not contain any lymphatic tissue. Follicular collections of lymphatic tissue are found only later in life, and in atrophic glands this tissue is absent.

Acute inflammatory changes were observed in the glands of five patients with diphtheria. In one case of leukemia there was bilateral enlargement of the glands as in the Mikulicz syndrome.

F. H. ADLER.

Lens

ROENTGEN-RAY CATARACT. P. J. LEINFELDER and H. I. KERR, *Am. J. Ophth.* 19:739 (Sept.) 1936.

From an experimental, clinical and microscopic study, Leinfelder and Kerr arrived at the following conclusions:

"1. Nonprogressive cataract was the usual result of irradiation of the rabbit lens with ordinary therapeutic doses of roentgen rays.

"2. In equal dosage short roentgen rays were better tolerated than the long.

"3. Two children, after a latent period of two years, developed opacities in the lenses following roentgen-ray therapy.

"4. No opacities occurred in the lenses of adults when the eye was shielded.

"5. Small doses of roentgen rays did not affect the lens (case 2, right eye).

"6. Nonprogressive changes consisted of a posterior polar horizontal linear opacity with radiating rows of vacuoles.

"7. Microscopic examination of rabbit and human lenses showed subcapsular swelling and degeneration of lens fibers.

"8. Primary injury by roentgen rays appeared to affect the lens epithelium, the cells of which subsequently formed pathologic lens fibers.

"9. Early microscopic changes were seen at the equator, but with growth the damaged fibers extended to the posterior polar region.

"10. Anterior subcapsular and cortical changes indicated more severe damage that resulted in total opacity of the lens.

"11. In the later stage of the nonprogressive cataract the microscopic changes were greatest at the posterior pole.

"12. Clinical and histological studies indicated that progressive cataract did not invariably follow exposure of the human lens to roentgen rays."

W. S. REESE.

STAPHYLOCOCCUS TOXIN COMBINED WITH LENS EXTRACT AS A DESENSITIZING AGENT IN INDIVIDUALS WITH A CUTANEOUS SENSITIVITY TO LENS EXTRACT. E. L. BURKY and H. C. HENTON, *Am. J. Ophth.* 19:782 (Sept.) 1936.

Burky and Henton report two cases which show that the cutaneous reaction to lens extract can be changed from positive to negative by the intracutaneous injection of a mixture of staphylococcus toxin and lens extract. This change cannot be effected by the injection of lens extract alone. The evidence also points to a loss of ocular sensitivity, though this was not proved.

W. S. REESE.

DINITROPHENOL CATARACT. H. F. WHALMAN, *Am. J. Ophth.* 19:885 (Oct.) 1936.

This article does not lend itself to abstracting. It is a review of the use of dinitrophenol in which the methods of use, experimental and clinical, are described. Whalman then describes the type of cataract

resulting from the use of this drug. A tabulation of data for twenty-seven cases of cataract as to the age and sex of the patient, the dose, the loss of weight, the time of onset after treatment and other data is given. Whalman states that the operative treatment of this type of cataract is successful, and for patients up to the age of 45 he favors linear extraction.

W. S. REESE.

CATARACT BRUNESCENS—STUDY OF THE NATURE OF THE COLORING SUBSTANCE. E. PUSCARIU and J. NITZULESCU, *Brit. J. Ophth.* 20: 531 (Sept.) 1936.

The authors have met with this form of cataract 4 times in 1,357 cases of cataract. In only one of the four instances was the patient myopic. In all four cases the postoperative course was perfectly normal. The vision after extraction of the cataract remained low, making the prognosis for such cataracts less favorable than that for ordinary senile cataract.

The two lenses of one patient were chemically examined. A colored substance could be extracted with hot 3 per cent solution of potassium hydroxide. All chemical tests made tended to prove that this substance belongs to the so-called "Abnützungspigment" (lipofuscin). The dopa reaction was negative. This finding does not exclude the possibility of an intermittent intervention of certain oxidative pigmentogenic enzymes, as advanced by Gifford and Puntenny.

Cataracta brunescens represents probably a far advanced stage of the process which produces usually yellow senile sclerosis of the lens. As in brown atrophy of the heart and of the muscles, it represents an accumulation of a pigment belonging to the group of lipofuscin.

W. ZENTMAYER.

POST-OPERATIVE DISTRESS IN CASES OF SENILE CATARACT. W. H. McMULLEN, *Brit. J. Ophth.* 20: 657 (Dec.) 1936.

McMullen states that postoperative distress in cases of senile cataract is partly physical and partly mental and that the physical and mental factors react one on the other. The physical causes are pain in the back and abdominal discomfort, usually associated with flatulence. The causes of the pain and distress are discussed. A specially designed mattress with a cushion beneath the small of the back and a pillow beneath the knees may delay the onset and lessen the severity of the pain in the back. The author has found it impossible to prevent or relieve the pain in many cases so long as the patient is kept flat on the back. The abdominal pain and flatulence may be relieved by diet and drugs, but no measure is so effective as permitting the patient to be propped up in bed at an early stage and to be turned on his side and allowed to draw up his knees.

To relieve mental distress the patient should be brought to the operating room in as confident and optimistic a mood as possible. Any technic which unnecessarily prolongs the operation should be avoided.

Two chief causes of postoperative mental distress are immobility and the binocular bandage. Restraint is unnecessary if the eyes are well

shaded and a watchful nurse is employed. A binocular dressing is applied for at least two or three days unless the patient finds it very trying, mental derangement occurs or conjunctivitis develops, in which case only the eye that was operated on is bandaged, and a flap of gauze is placed over the other eye, which the patient can lift when desired. If no complications arise the patient is kept in bed only two or three days.

The article is well worth perusal by all ophthalmic surgeons.

W. ZENTMAYER.

Lids

OPERATIVE TREATMENT OF SPASMODIC ENTROPION. N. POCHISOW, *Ann. d'ocul.* 173:737 (Sept.) 1936.

An article on the operative treatment of spasmodic entropion by Poulard, published in the *Annales d'oculistique* in February 1935, was similar to an article by Pochisow published in January 1935 in the *Sovetskiy vestnik oftalmologii*. As Pochisow's method differs somewhat from the operation of Poulard he has thought it wise to refer again to this subject.

The operation consists of two parts: classic canthotomy and section of the external ligament lengthwise (the length of the tendon). It is important to obtain section of the ligament from the point of insertion at the external border of the orbit. Pochisow now does canthoplasty and uses two methods.

In the first method he places the sutures of the lower lid in such a way as to stretch it, by drawing upward, toward the palpebral conjunctiva. The second suture is placed so as to enter in a similar way and to cause an artificial entropion, which disappears in a few days, the lid becoming normal. In the second method the needle is introduced into the skin of the upper lid, the conjunctiva not being touched, and passed into the conjunctiva of the lower lid near the end of the cul-de-sac. It finally passes into the skin of the lower lid at a little higher level. By drawing the palpebral conjunctiva higher and outside and fixing it in this manner one prevents the possibility of recurrence.

There are three illustrative drawings.

S. H. McKEE.

SURGICAL TREATMENT OF CONGENITAL COLOBOMA OF THE LIDS. J. CHARAMIS, *Ann. d'ocul.* 173:810 (Oct.) 1936.

Congenital coloboma of the lids, a rare malformation, was first described at Montpellier in a thesis of Mayer in 1808. A more thorough study was published for the first time in England by Wylde in 1862. Interesting are the reports by von Hippel in 1907 and Pagenstecher in 1912. Among recent reports the writer cites that of van der Hoeve in 1921.

A general description of coloboma and the different theories to account for it are given. Charamis describes a case in which he had followed the patient from birth. A girl was examined for the first time at the age of 6 months and again at the age of 9 years; no treatment had been carried out. The father had syphilis, and the child was given antisyphilitic treatment. There were no other congenital anomalies. The cornea was intact, although it was completely uncovered with the lids closed, so it was continually exposed and without protection.

The coloboma occupied nearly the whole width of the upper lid and extended practically to the limits of the tarsus. However, the movements of the upper lid were conserved despite this.

As regards the etiology, Charamis thinks that syphilis should be taken into serious consideration. This accords with the theory of van Duyse. The treatment of all palpebral colobomas should be surgical. Sometimes a simple drawing together of the border suffices; for those of greater width blepharoplastic operations are necessary. The correction in the case observed by Charamis is described in detail, and the article is illustrated with five photographs.

S. H. MCKEE.

Methods of Examination

AN ILLUMINATING DEVICE TO BE USED AS AN ATTACHMENT TO THE BINOCULAR CORNEAL MICROSCOPE FOR GONIOSCOPY AND GONIO-PHOTOGRAPHY. R. CASTROVIEJO, *Am. J. Ophth.* 19:786 (Sept.) 1936.

This article describes an illuminating device that can be attached to a corneal microscope so as to permit gonioscopy, goniophotography and examination of the fundus by means of contact glasses. Photographs of the fundus were also obtained by this method.

W. S. REESE.

INFRA-RED PHOTOGRAPHY OF THE FUNDUS OF THE EYE. E. DIAZ-CANEJA, *Bull. Soc. d'opht. de Paris*, January 1936, p. 55.

The history and evolution of the use of red-free light and of the infra-red rays in ophthalmology and photography are reviewed. Nineteen infra-red photographs are included in the article, and a description is given. A reading of the entire work is necessary to understand the article clearly.

L. L. MAYER.

Neurology

EMOTIONAL FACTORS IN MENTAL RETARDATION: A READING PROBLEM. R. C. HAMILL, *Arch. Neurol. & Psychiat.* 36:1049 (Nov.) 1936.

Hamill considers the emotional factor in reading difficulties, summing up his point of view in the following conclusion: "The fear of words can lead to evasions that interfere with learning to read. Looking ahead to avoid trouble may lead to difficulties with recognition and pronunciation of the word at hand. This may have some relationship to such mechanisms as reversal. Children, not knowing words, can be led into fears of meeting taboo words. Such fears lead to resentments and rejections of learning. Most taboo words have to do with natural processes of the body. Desire to know about these processes is inevitable. Expressions of that desire are frowned on. Words are the natural means of expression. Knowledge is feared and resented." Two cases of reading disability are cited in which Hamill believes the emotional element was sufficient to cause the difficulty. He also discusses the reading problem in relation to handedness and attempts to show a relationship between handedness and emotional conflicts.

R. IRVINE.

Ocular Muscles

THE ACTION OF THE OCULAR MUSCLES. J. VAN DER HOEVE and C. O. ROELOFS, *Arch. f. Augenh.* 110: 1, 1936.

Van der Hoeve and Roelofs used Volkmann's data on the origin and insertion of the ocular muscles with certain corrections, and with the aid of a coordinate system have calculated the position of the origin and insertion of each of the ocular muscles in relation to the point of rotation of the eye. This point lies 1.29 mm. back of the center of the eye. Although these values are relative, they give information regarding the action of each of the ocular muscles and their axes of rotation.

The results prove that the superior oblique muscle and the inferior oblique muscle are mutual antagonists and that for every ocular movement at least three ocular muscles come into play.

F. H. ADLER.

Operations

SOME OCULAR OPERATIONS. M. AMSLER, *Schweiz. med. Wchnschr.* 66:1268 (Dec. 12) 1936.

The three procedures discussed are dacryocystorhinostomy, extraction of the cataract in its capsule and the operations for detachment of the retina. The West technic is reserved for the rhinologist. The operation of Toti as modified by Dupuy-Dutemps and Bourguet is for the ophthalmologist. Its indications are precise. It is limited to the correction of the obstruction of the nasolacrimal canal caused by chronic dacryocystitis. It aims to provide catheterization of the canal, removal of diseased tissues and restoration of function. It can be performed in forty-five minutes or less.

By extracting the lens in its capsule there is no necessity for awaiting the maturity of the cataract, and discission of the secondary membrane is eliminated.

Detachment of the retina was considered an incurable condition until the time of Gonin. Closure of the tear or hole is of the utmost importance to secure a successful result. The Paquelin cautery has been abandoned as an instrument of historical interest only. Galvanocautery, electrolysis and diathermy are now the technics of choice.

Ocular surgery has made rapid strides in recent times.

L. L. MAYER.

Orbit, Eyeball and Accessory Sinuses

A CASE OF "EXOPHTHALMIC OPHTHALMOPLÉGIA WITH THYROTOXICOSIS." H. B. STALLARD, *Brit. J. Ophth.* 20: 612 (Nov.) 1936.

A case of exophthalmos occurring in conjunction with unilateral partial ophthalmoplegia affecting the inferior rectus and inferior oblique muscles and associated with a basal metabolic rate of —4 per cent and mild general symptoms of thyrotoxicosis in a man 31 years of age is reported. After the administration on three successive days of 600 organon units (1,200 Schoeller units) of thyrotropic hormone there were a temporary rise in the basal metabolic rate, an increase in the

temperature and in the pulse and the respiration rate, increased exophthalmos, digital tremor and a perspiring, clammy skin. Later, despite tarsorrhaphy, it was necessary to enucleate the eye because of extensive destruction of the cornea.

The extra-ocular muscles were five or six times the normal size and were pale and fusiform. Histologically the muscle tissue showed interstitial fibrosis and chronic inflammatory infiltration by round cells. The muscle fibers exhibited no definite degenerative changes.

The clinical features of the case just reported—the relatively small degree of exophthalmos, the moderately increased pulse rate (from 64 to 90), the slight enlargement of the thyroid and the markedly defective action of the two extra-ocular muscles of the left eye on the same side as the greater degree of exophthalmos—conform to those described by Russell Brain under the diagnostic label of exophthalmic ophthalmoplegia thyrotoxicosis. These features, together with the low basal metabolic rate (—4 per cent) and the relatively slight general symptoms, suggest that possibly some factor other than the products of hyperactivity of the thyroid gland was responsible for the exophthalmos and the changes in the extra-ocular muscles. "The injection of thyrotropic hormone of the pituitary was in the nature of a clinical test and it produced such interesting events as a temporarily raised basal metabolic rate, increased temperature, pulse and respiration rate, increased exophthalmos, digital tremor and a perspiring, clammy skin. It may be that the degree of exophthalmos which developed after these injections of thyrotropic hormone would have been less in a case of a normal control."

W. ZENTMAYER.

PATHOLOGIC OCULAR HYPOTENSION. A. MAGITOT, *Ann. d'ocul.* 173: 785 (Oct.) 1936.

In spite of imperfections the tonometer may be used for taking the normal tension, a high and a low limit being recognized. The normal tension is approximately 10 mm. of mercury, but one must not consider this an absolute standard. In other words, the ocular tension must always be compared with the general arterial pressure.

There are three influences which modify the ophthalmic tension: the local circulation, substances circulating in the blood and nervous influences. These influences may be found singly, but oftener they are combined.

In certain cases hypotension is due to congenital malformations; in others it is due to familial myopia, but hypotension in myopia is not the rule. Among the traumatic hypotensions are those that follow trauma in the ciliary region, burns of the limbus and contusions and those that are caused by surgical intervention on the globe. Hypotension in detachment of the retina is far from constant. It is frequent and does not appear to be linked to the myopia, and when it does appear it causes profound disorganization of the ocular membranes. In infections and toxemias, hypotension is frequent and here one meets the problem of hormonal influences.

Nervous influences constitute an important factor in hypotony, but what has been called transitory or essential ophthalmomalacia has become rare since the tonometer has come into use.

S. H. MCKEE.

RESTORATION OF THE EYE SOCKET WITH A THIERSCH GRAFT. LIN CHING-K'UEI, Chinese M. J. 50: 1335 (Oct.) 1936.

The author describes in considerable detail six cases in which complete or partial plastic restoration of the eye socket was attempted. In five cases this was successful, and in one case failure resulted. The pre-operative requirements, such as correction of any deformity of the lid, the elimination of pus-forming pockets and treatment of the margins of the lid, are stressed. As an anesthetic procaine hydrochloride, given by retrobulbar injection, was used.

The operation, the preparation of the mold and the taking of the graft are described in detail. No mention is made of the work of either Esser or Gillies.

The article is profusely illustrated, and the importance of plastic surgical procedures on the eye in China is emphasized.

S. H. MCKEE.

The Pupil

PUPILLARY VARIABILITY IN ONE HUNDRED AND EIGHT SYPHILITIC PATIENTS. T. M. SHAPIRA and F. M. CRAGE, Am. J. Ophth. 19: 891 (Oct.) 1936.

Shapira and Cragge discuss the significance of the Argyll Robertson pupil and anisocoria. They give the following summary:

"In this series of 108 syphilitic patients, 32 patients had pupillary irregularity in both eyes; in 8 patients the pupils were irregular in one eye.

"Pupils were unequal in 42 patients.

"Pupils did not react to accommodation in 19 patients.

"Reaction to light was absent in 33 patients.

"Definite typical Argyll Robertson pupils were present in 17 patients.

"Five patients showed reflex pupillary rigidity.

"Considering a miotic pupil as being less than 2.5 mm., miosis was present in both eyes in 15 patients, and in one eye in 16 patients."

W. S. REESE.

Physiology

DISTRIBUTION OF INTENSITY OF THE LIGHT SENSE IN THE PERIPHERAL PARTS OF THE RETINA. L. N. GASSOWSKY and N. J. NIKOLSKAYA, Sovet. vestnik oftal. 9: 123, 1936.

Gassowsky and Nikolskaya measured in sixteen subjects the absolute threshold for light of the periphery of the retina and the corresponding sensitivity of the retina at separate points (inverse to the threshold) on both sides of the fovea, in the horizontal meridian. The threshold for light was determined for point stimuli, with a visible angle of 2 minutes and 28 seconds at 4, 6, and 8 degrees and up to 20 degrees from the fovea. The basic parts of the experiment were white and red illuminated dots which could be seen by the observer through the slit of a screen; the intensity of their illumination could be regulated by

the observer. The observer remained in complete darkness for one hour and a half before he could start registering his findings with the illuminated dots. Further details of the experiment are given in detail.

The authors illustrate with a table and diagram that the absolute threshold for light is decreased up to 10 degrees from the fovea on both the nasal and the temporal side. The sensitivity of the retina at 10 degrees from the fovea is twice as great as at 4 and 20 degrees.

O. SITCHEVSKA.

Retina and Optic Nerve

INHERITANCE OF OPAQUE NERVE FIBRES IN THE RETINA (PAPILLA LEPORINA). E. A. COCKAYNE, *Brit. J. Ophth.* 20: 569 (Oct.) 1936.

After describing the development of opaque nerve fibers and enumerating the various congenital anomalies which have been found associated with this condition, Cockayne gives the pedigree of a family in which ten members are affected: In four of them the condition is bilateral; in six, unilateral. In all of them the area covered by the opaque fibers is small. Five males and five females are affected, and in two of each sex the condition is bilateral. Of those examined in the sibships containing affected members, ten (five males and five females) are affected, and ten (four males and six females) are normal. Thus the ratio of affected to normal persons is 1:1, and descent is direct in every case. Males and females show the condition to an equal extent, and it is bilateral in the same number of females as of males. Both these observations are contrary to expectation if it is sex-linked. It is true that there is no instance of transmission of the abnormality by a male, but only one of the affected males has children, and only one of these children is old enough to show the defect.

Though the pedigree does not disprove Kisö's hypothesis that the condition is sex-linked, its inheritance is quite in accord with that of autosomal dominants, and since autosomal dominants are common and sex-linked dominants are of extreme rarity in man it is probable that opaque nerve fibers fall into the former rather than into the latter group.

W. ZENTMAYER.

BIPOLAR ELECTROLYSIS IN THE TREATMENT OF DETACHMENT OF THE RETINA. HUDELO, *Bull. Soc. d'opht. de Paris*, October 1935, p. 642.

Electrolysis for the treatment of detached retina is not new, especially the unipolar method, which was used before 1895 by Abadie, Gillet de Grandmont, Moll, Terson and, more recently, by Vogt. The bipolar method was employed in 1893 by Schoeler, but his technic was entirely different from the original simple and efficacious method of Hudelo.

The objection to the unipolar method is that it coagulates only at the level of the electrode in a single point, and the electrolysis is limited to feeble intensities in order not to damage the vitreous. The bipolar method fixes the globe and coagulates an area in the form of a crown. Platinum-iridium electrodes separated and insulated from each other at a distance of 3 mm. may be used without complete removal of the

conjunctiva over the area as done by Arruga. The entire technic is described. Adequate ophthalmoscopic control is always present. Five observations are reported.

L. L. MAYER.

FIVE OBSERVATIONS OF TRAUMATIC LESIONS OF THE MACULA. C. COTELA, H. LAGRANGE and L. BONHOMME, Bull. Soc. d'opht. de Paris, December 1935, p. 804.

The authors report in detail five cases in civilians in which trauma caused lesions of the macular area, and compare these traumas with traumas occurring during the war period. The observation that any type of trauma to the region of the head may cause macular lesions, as this area is seemingly less resistant than others, is in accord with the findings of F. Lagrange during the war period. Delay in finding macular lesions is common and therefore has an important medicolegal interest.

L. L. MAYER.

LOCAL SURGICAL TREATMENT OF PAPILLARY STASIS. GOMEZ-MARQUEZ, Bull. Soc. d'opht. de Paris, December 1935, p. 814.

A surgical method to prevent the occurrence of atrophy of the optic nerve due to edematous pressure in papilledema is presented. Canthotomy is done on the temporal side, and then a vertically curved incision enables one to make an exposure whereby, when the external rectus muscle is divided, the optic nerve is readily made accessible. Tenon's sheath and connections with the dura are slit by the assistance of an aneurysm needle. Gomez-Marquez has practiced the operation on cadavers but has not used it as yet on living human subjects. Excellent drawings depicting the procedure accompany the article. It is believed that the chances of infection of the brain are much less than with a cranial operation and that such an operation may be especially useful in cases of increased intracranial pressure in which localization is not exact or in which a more formidable operation is not warranted.

L. L. MAYER.

NEW PATHOGENIC AND THERAPEUTIC TRENDS IN TABETIC ATROPHY OF THE OPTIC NERVE. H. ARRUGA, Arch. de oftal. hispano-am. 36: 365 (July) 1936.

This paper is based on Arruga's clinical experience in connection with Lauber's theories on tabetic atrophy of the optic nerve. These concern the general arterial hypotony and the relatively increased intra-ocular tension in this condition. The indicated treatment is raising the general tension and lowering the intra-ocular tension. Although the number of cases in which Arruga applied the treatment was fifteen, he reports only six, the others having been observed too short a time or incompletely. Among these a fairly satisfactory result was observed in three. Apart from the antisiphilitic treatment, general treatment consisted of dietetic measures and the use of strychnine, caffeine, epinephrine and ephedrine; the local treatment consisted in the administration of pilocarpine and, occasionally, cyclodialysis.

C. E. FINLAY.

DIAGNOSTIC AND PROGNOSTIC VALUE OF EXAMINING THE BLOOD VESSELS OF THE EYE IN HYPERTENSION. N. A. PLETNEVA, *Sovet. vestnik oftal.* 9: 3, 1936.

Hypertension, according to Pletneva, is on a neurohumoral basis; it is a form of the functional vegetative neurosis; a spasm of the precapillary system is produced, which leads later to sclerotic changes in the precapillary vessels. Pletneva examined 104 patients with hypertension in the Institute of Functional Diagnosis. The ages of the patients varied from 20 to 80 years. The systolic blood pressure in all the patients was above 170 mm. of mercury. Not only the retinal but also the conjunctival and limbal blood vessels were examined. Eighteen patients suffered from albuminuric retinitis with spasm of the blood vessels. Seventeen of these patients had nephrosclerosis, and ten of them died. Eight patients had spasm of the retinal vessels; the arteries were narrow, some appearing like silver wires, with a marked difference in caliber between the arteries and the veins. Ten patients suffered from unilateral thrombosis of the central vein without the kidneys being affected. Salus' symptom (indentation of veins by arteries at the crossing) was observed in 50 per cent of the patients. Thirty patients showed this symptom while the kidneys were unaffected. The so-called angiospastic retinitis of Volhard is met with in "pale" hypertension (i. e., there is a spasm of the peripheral vessels), which indicates a pathologic condition of the precapillary system of the peripheral vessels, the prognosis of which is poor.

With the slit lamp Pletneva examined the capillaries of the corneal limbus in 91 patients. Twenty-eight patients suffering from "pale" hypertension presented complete obliteration or absence of the fine limbal net. In some capillaries which retained their patency an engorgement of the venous knee could be seen. The blood circulation was irregular. Nineteen of these patients showed capillary changes coinciding with those of the retinal vessels. All these patients suffered from nephrosclerosis. In "red" hypertension (i. e., the peripheral vessels were dilated) the limbal capillary net was engorged and hyperemic; several aneurysms and hemorrhages were noted.

Pletneva draws the following conclusions: The prognosis of hypertension can be made by careful examination of the retinal vessels and of the capillaries of the corneal limbus. Angiospastic retinitis is usually met with in patients who have sclerosis of the blood vessels of the kidneys, brain and heart; it is prognostic for malignant hypertension. An ophthalmologic examination should always be made in hypertension, as the changes in the blood vessels of the eye indicate the condition of the peripheral blood vessels of other organs: heart, brain and kidneys.

O. SITCHEVSKA.

Trachoma

TREATMENT OF TRACHOMA IN CHILDREN. M. ZACHERT, *Rev. internat. du trachome* 13: 177 (Oct.) 1936.

Trachoma in children is generally benign and is rarely associated with severe complications of the cornea or lid. Though children have a lesser immunity to the contagion than adults, their resistance to its

progress is greater. The granulations should be crushed individually and in several sittings. Frottage with chaulmoogra oil is well tolerated.

J. E. LEBENSOHN.

TRACHOMA THERAPY WITH QUININE BISULPHATE. E. SELINGER, Chinese M. J. 50: 1449 (Oct.) 1936.

When applied to mucous membrane, quinine bisulfate acts as an astringent, is mildly anesthetic and, being an alkaloid, penetrates through the mucous membrane. It is a bactericide, and because it is a protoplasmic poison it destroys leukocytes and lymphocytes and inhibits the invasion of the tissues by these elements. The bisulfate is used because it is more water-soluble than most of the other salts.

The results obtained indicate that this form of treatment is shorter than that with copper sulfate and chaulmoogra oil, that corneal complications are favorably influenced by it and that the final visual results are better than those obtained with other forms of therapy for trachoma.

S. H. McKEE.

Tumors

PAPILLOMATA AND OTHER TUMORS OF THE LIDS AND THEIR TREATMENT WITH THE ELECTROLYSIS NEEDLE. J. MINTON, Brit. J. Ophth. 20: 624 (Nov.) 1936.

This is a plea directed to the ophthalmic surgeon for the use of the electrolysis needle for the removal of certain tumors of the lids.

The galvanic current is required. The machine now used in the epilation of lashes may be employed. A curved or straight steel needle is preferred. A current of from 3 to 4 milliamperes is necessary for destruction of the base. In the removal of a papilloma the needle is made to transfix the growth in several directions. The needle is kept in position at each transfixion only a few seconds. The growth is then snipped off above the cauterized base. If the needle is left in too long or if the current is too strong the surrounding healthy tissue will be destroyed and a scar may result. Other growths require certain modifications of the technic just described.

W. ZENTMAYER.

TUMOR OF THE OPTIC NERVE (NEUROPERINERVOSA GLIOMATOSA). F. ROSSEAU, A. DOLLFUS and G. ARDOIN, Bull. Soc. d'opht. de Paris, October 1935, p. 629.

For the past two years it had been noted that a boy of 7 years had prominence of his right eye. From time to time there had been a spontaneous receding to normal. The exophthalmos was not reducible; excursion was nil upward and was limited in all other directions, mostly laterally. The lids could be closed, and the cornea was well protected. The papilla showed stasis, with atrophy of the superior portion of the optic nerve. Pupillary reaction was absent, although the patient counted fingers at 30 cm. The sinuses were not involved. The tumor is pictured grossly, and several photomicrographs show the nature of the tissue cells. The tumor entirely surrounded the optic nerve.

L. L. MAYER.

CONGENITAL PIGMENTED CYSTS OF THE IRIS AND THE CILIARY ZONE; VESTIGE OF THE VASCULAR MEMBRANE OF THE LENS; PERSISTENCE OF THE HYALOID ARTERY. F. BADEAUX, Bull. Soc. d'ophth. de Paris, January 1936, p. 68.

The clinical rarity of congenital pigmented cysts of the iris and the ciliary zone and the infrequency of reports of the condition, as studied by means of the slit lamp or by pathologic examination, prompted this article. In a patient 10 years of age, on dilatation of the left pupil a mass was found back of the iris between 6 and 9 o'clock. Pictures taken with the use of the slit lamp are reproduced. The history of such cysts is reviewed, and drawings are shown to demonstrate the embryology of this type of cyst. Badeaux believes that such cysts are caused by doubling of the epithelium of the uveal layer and subsequent incomplete absorption. Surgical intervention is indicated if visual acuity is lowered. If such a growth is watched over a long period its benign nature is confirmed.

L. L. MAYER.

Uvea

UVEITIS ASSOCIATED WITH ALOPECIA, POLIOSIS, DYSACOUSIA AND VITILIGO. T. H. LUO, Chinese M. J. 50: 1409 (Oct.) 1936.

The type of uveitis described is bilateral, develops rapidly and reaches its height in a short time. Often it is preceded by such prodromal symptoms as headache, drowsiness, nausea and a feeling of heaviness about the head. In most of the Japanese reports, lesions of the choroid are reported. Late in the course of the disease the ocular picture is chiefly that of inflammation of the anterior part of the uvea, with the formation of seclusio and occlusio pupillae, which prevent ophthalmoscopy and may conceal changes in the fundus.

The alopecia and poliosis which occurred in practically all the reported cases made their first appearance about from three weeks to nine months after the onset of the disease. The vitiligo, dysacusia and tinnitus were present in a little over half the cases. Tinnitus with temporary impairment of hearing was noted early in the course of the disease, while vitiligo appeared shortly after the development of the alopecia and poliosis.

The etiology of the disease is unknown; consequently the treatment is nonspecific. The grave prognosis should always be borne in mind.

S. H. McKEE.

Vitreous

CLINICAL STUDIES ON THE VITREOUS: THE DETACHMENT OF THE CHOROID OF LEAKING EYES. K. LINDNER, Arch. f. Ophth. 135: 462 (July) 1936.

If the aqueous is aspirated from the eye of a cadaver, a fluid which is obviously derived from the vitreous slowly refills the chamber. The vitreous gives off this fluid under the influence of pressure created by the forward displaced lens. The same mechanism, according to Lindner,

applies to the living eye. In a leaking eye (with a corneal or vitreous fistula) no such pressure is available. In leaking eyes with choroidal detachment the volume of the vitreous is greatly reduced; it must have given off water, not under the influence of any pressure, but under the influence of chemical forces. The regenerated, abnormal aqueous which seeps through the vitreous in these fistulating eyes contains the agent that makes the vitreous shrink. This shrinking is the real cause of choroidal detachment. In the eyes of young persons the vitreous detaches itself from the retina more easily than in the eyes of older persons. That is the reason why choroidal detachment occurs only in older persons. (See also Lindner: *Arch. f. Ophth.* 135: 332, 1936.)

P. C. KRONFELD.

ANATOMIC STUDIES OF POSTERIOR SEPARATION OF THE VITREOUS. L. SALLMANN, *Arch. f. Ophth.* 135: 593 (July) 1936.

Lindner's views concerning the mechanism of retinal detachment and the clinical observations of posterior separation of the vitreous made with the slit lamp by those belonging to Lindner's school have brought the anatomy of this condition to attention again after von Hippel in 1908 had practically settled the until then very active discussion concerning it. Sallmann has examined anatomically twelve fresh human eyes with various clinical conditions, studying them shortly after enucleation and without the use of a fixative. In two of these eyes posterior separation of the vitreous was macroscopically visible; in two others macroscopic combined with histologic examination made the diagnosis of the condition certain. The usual fixing and hardening process often produces artificial posterior separation, in which case not a trace of albuminous fluid can be found between the retina and the separated vitreous. A true separation is exaggerated by the fixing process. To differentiate between the hyaloid membrane and a border layer produced within the vitreous by partial liquefaction, the pyridine method of Redslob is very useful, because it produces a well defined opacity of the hyaloid membrane only.

P. C. KRONFELD.

Sympathetic Ophthalmia

A CASE OF SYMPATHETIC OPHTHALMIA FOLLOWING GONORRHEAL CORNEAL ULCER, OBSERVED FOR NINETEEN YEARS. GROENOUW, *Klin. Monatsbl. f. Augenh.* 96: 742 (June) 1936.

Groenouw refers to Peters' report in the literature for 1919-1935 on sympathetic ophthalmia and describes the case of a man aged 43 who suffered a rupture of a gonorrheal corneal ulcer and prolapse of the iris in 1911. The second (right) eye became inflamed two months later; the irritation disappeared after local treatment and enucleation of the left eye. However, recurrences were observed after one, seven, eight and nine years and in a mild form thereafter. The eye was found to be free from any symptoms, and vision at the last examination, in 1930, was practically normal. Sudden death followed soon after this examination. The blood count, which is supposed to show changes indicative of sympathetic ophthalmia, showed a decided decrease of polymorpho-

nuclear leukocytes and an increase of eosinophils and mononuclear lymphocytes. Small, round, white dots, characteristic of sympathetic choroiditis, were observed in the peripheral portion of the choroid. Groenouw's case is the third case of this type in which these dots disappeared. The author stresses the fact that there were numerous recurrences, although recovery was promptly obtained in the beginning, and that nevertheless the condition of the eyes allowed strenuous visual activity.

K. L. STOLL.

Therapeutics

GLYCEROL TRINITRATE (NITROGLYCERINE) IN THE TREATMENT OF HEMERALOPIA (NIGHT-BLINDNESS). R. DE R. BARONDES, Brit. J. Ophth. 20: 528 (Sept.) 1936.

To the usual condition to which hemeralopia is ascribed, Barondes adds a circulatory disturbance of dysfunction in which the retinal arteries and capillaries remain in a spastic state. This spasticity is observed to be more pronounced in the vessels in the fringe of the retina, that part of the retina under faint illumination. The nerve endings in the light-perceiving area—the retinal rods—are dependent on a proper supply of food for the regeneration of the visual purple, the “night-seeing agent.”

Barondes gives the ophthalmic finding in five adults with hemeralopia as a constricted, blanched appearance of the peripheral portions of the retinal arteries, with more or less venous hyperemia and congestion.

Nitroglycerin proved to be the most effective dilator of these spastic vessels, although the nitrites gave almost comparable results. It is suggested that vasodilator drugs might be administered to those working more or less continuously in the dark, to whom it is especially important to be able to see in darkness, as spies in wartime, navigators and those aviators who do night flying.

W. ZENTMAYER.

HYPODERMIC INJECTIONS OF CAROTENE IN THE TREATMENT OF PHLYCTENULAR CONDITIONS AND VITAMINE A DEFICIENCY DISEASES IN OPHTHALMOLOGY. M. C. TAI, Chinese M. J. 50: 1453 (Oct.) 1936.

Cod liver oil possesses an unpleasant smell and is difficult to preserve from oxidation. Eggs, milk and butter are sometimes not well digested by certain patients. Vegetables are seasonable products and, even when available, may cause gastro-intestinal upsets. Carotene in ampules, administered hypodermically, obviates all the difficulties of oral ingestion. The only contraindication to its use is cirrhosis of the liver. Rigid asepsis should be observed in administering it, otherwise a moderate degree of dermatitis may develop. Carotene is inexpensive and is available in large amounts. It is produced in China and is therefore easily obtained in that country and may be widely used to great advantage in treating certain ocular conditions there.

S. H. McKEE.

News and Notes

SOCIETY NEWS

Annual Congress of the Ophthalmological Society of the United Kingdom.—The following program was presented at the annual congress of the Ophthalmological Society of the United Kingdom, held from April 29 to May 1, 1937:

"Prognosis in Papilledema," Dr. Gordon M. Holmes, president.

"Retinal Circulation, Changes in Metabolic Disease," Dr. A. E. MacDonald, Toronto, Canada.

"Some Observations on Katholysis in Treatment of Retinal Detachment," Mr. R. Foster Moore.

"Criticisms and Comments on Newer Methods of Treating Detachment of the Retina," Mr. J. Cole Marshall.

"The Internal Limiting Membrane of the Retina," Mr. E. Wolff.

"Importance of the Near Balance Test," Miss Margaret Dobson.

"Pathogenesis of the Melanomata," Mr. E. F. King.

"Principles and Practice of Ocular Screening," Mr. G. F. Alexander.

"Clinical and Microscopic Appearances of Experimental Grafts of Scleral Tissue onto a Rabbit's Cornea," Mr. J. W. Tudor Thomas.

"Atropin Irritation," Mr. Fred Ridley and Dr. Ian Maclean.

"Innervation of Ocular Muscles and the Mesencephalic Root of the Fifth Nerve," Prof. H. Woollard.

"Spontaneous Cure of Retinal Glioma," Mr. Montague Hine.

"Visual Purple," Dr. R. J. Lythgoe and Dr. C. F. Goodeve.

"Exophthalmic Ophthalmoplegia," Dr. W. Russell Brain.

"Exophthalmic Ophthalmoplegia: Effect of Prostigmin in Two Cases," Prof. F. R. Fraser.

"Case of Exophthalmic Ophthalmoplegia," Mr. H. B. Stallard.

"Involvement of the Optic Nerve in the Demyelinating Diseases," Dr. J. Purdon Martin.

There was also an open discussion on the rarer forms of keratitis.

The president of the society and the medical staff of the National Hospital for Diseases of the Nervous System arranged a demonstration of cases of ocular palsies, disturbances of the visual field, ophthalmoscopic conditions and pathologic specimens of neurologic and ophthalmologic interest.

The following officers were elected for the year 1937-1938: president, Gordon M. Holmes; vice-presidents, T. Harrison Butler, R. Affleck Greeves and Percival Hay; treasurer, Sir Arnold Lawson; council, O. Gayer Morgan, Sir Stewart Duke-Elder, Ida Mann, A. Macrae, R. C. Davenport and J. D. M. Cardell; secretaries, H. B. Stallard and L. E. Savin.

GENERAL NEWS

Summer Courses in Ophthalmology and Otolaryngology in Vienna, Austria.—The twelfth intensive European summer courses in ophthalmology and otolaryngology, under the direction of Dr. George

W. Mackenzie, will be given in Vienna, Austria, during the summer of 1937. The course in ophthalmology includes microscopic anatomy and pathology of the eye; external diseases of the eye, with an exhibition of interesting cases; ophthalmoscopy, with an exhibition of cases; ward work demonstrating the treatment of patients, including the postoperative treatment; operations on pigs' eyes, and courses in the ocular muscles and slit lamp examination. The group leaves New York on July 10 and returns on September 4. Further information may be secured from Dr. Mackenzie, 269 South Nineteenth Street, Philadelphia.

CORRECTIONS

In the article by Prof. A. Bielschowsky entitled "Application of the After-Image Test in the Investigation of Squint," which appeared in the March issue (17: 408, 1937), the diagram in the last line but one

on page 418 should have been $\begin{array}{c} | \\ \text{---} \\ | \\ \text{L} \end{array}$ — — R. And the word "crossed," at the

end of the fourth line on page 419, should read "uncrossed."

In the article by Dr. Samuel P. Oast, "Scleromalacia Perforans: Report of a Case," in the April issue (17: 698, 1937), two errors occurred. In the sixth line on page 700, "lymph nodes" should be inserted after "preauricular," and in the twentieth line on the same page, "eye" should be omitted after "neither."

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Jan. 18, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

DACRYOCYSTORRHINOSTOMY—TECHNIC AND RESULTS. DR. LOREN P. GUY.

A brief history of dacryocystorhinostomy from 1724 to the present was given. The indications, contraindications and complications were enumerated. A simple technic, requiring less than thirty minutes to perform, was described. Of the first 15 cases in which this operation was performed, success was reported in 13.

DISCUSSION

DR. WEBB W. WEEKS: Of all the procedures used to accomplish drainage of tears, the one that accurately approximates the edges of the wound and leaves the tissues in normal relationship seems the correct surgical operation. Dupuy-Dutemps and Bouget not only insisted on the contraindications mentioned in Dr. Guy's report, particularly the patency of the puncta and canaliculi, but also emphasized that the operation should be done when inflammation of the tissues was absent.

Since my report in 1932 of 8 cases in which the procedure previously described was carried out, it has been done in 6 additional cases. During the past year Dr. Wendell Hughes has carried out this procedure on 7 patients in our service at the New York Eye and Ear Infirmary. In 1 case the edges of the wound could not be sutured because of friable tissue. In another case the lower one third of the sac was carried through the mucous membrane of the nose and left in situ. All these patients have done well except 1, in whom the lower canaliculus was partially slit some years previously, with resultant impairment of function of this lacrimal drainage system.

According to the French procedure anesthesia is produced by the subcutaneous injection of a 2 per cent solution of procaine hydrochloride plus a 1 per cent solution of neosynephrin hydrochloride along the course of an incision in the skin extending to the dome of the lacrimal sac above and to the neck of the lacrimal sac below. In addition, the branch of the nasociliary nerve passing through the anterior ethmoid foramen is blocked, and the mucous membrane in front of the middle turbinate is raised by the same solution. The latter step protects the nasal mucous membrane during the removal of bone.

ANISEIKONIA WITH NO REFRACTIVE ERROR. DR. WENDELL L. HUGHES.

The relative size of the extra-ocular images in a group of 43 patients with symptoms that were thought to be ocular in origin was determined. In 14 the images were equal. In 29 they were asymmetrical.

The result in 14 cases in which complete data were obtainable was reported. Relief was obtained in 10 by the wearing of iseikonic zero power lenses designed to correct the aniseikonia present. It has been shown that the presence of aniseikonia is independent of any refractive error.

DISCUSSION

DR. JOSEPH I. PASCAL: I noticed that about 91 per cent of one group of patients with aniseikonia and photophobia were relieved by iseikonic lenses. It is known that ordinary white glass absorbs about 10 per cent of the light. Is there any possibility that the photophobia was largely relieved by the reduction of this 10 per cent of the light?

DR. WENDELL L. HUGHES: The question of photophobia is an interesting one, and I do not know exactly how to explain it, but the 91 refers to the percentage of patients complaining of photophobia who had aniseikonia. Most of the patients had been wearing lenses of minor strengths previously, and the photophobia had been unrelieved. The photophobia was relieved when the images were made symmetrical in size and were able to be fused easily.

CATARACT DUE TO DINITROPHENOL. DR. CLYDE McDANNALD.

A case of cataract due to dinitrophenol was reported. The patient complained postoperatively of blurred vision, which was overcome by squinting. Transillumination demonstrated scattered areas of atrophy of the pigmented layer of the uvea. There were similar small rarefied areas in the eye on which no operation was performed. It was suggested that the rarefaction of the uveal pigment may be a factor in a toxemia causing opacities of the lens after the ingestion of dinitrophenol.

USE OF CELLULOSE ADHESIVE TAPE FOR RETAINING DRESSINGS FOR THE EYE. DR. DONALD BOGART.

A cellulose adhesive tape has been used at the New York Eye and Ear Infirmary for eighteen months for approximately 2,500 patients. It has the following advantages:

1. It retains dressings for the eye in place well and is easily removed.
2. It is less disfiguring to the patient than ordinary adhesive tape.
3. It is available in a large dispenser, which allows the cutting of pieces with one hand, thus promoting speed.
4. It is very much less expensive than ordinary tape used for dressings for the eye.
5. It usually leaves no residue on the face.
6. It is waterproof.

Scotch tape has the following disadvantages:

1. In warm weather the small roll may be difficult to "start."

2. In 5 of the 2,500 patients a mild dermatitis due to the tape developed. Patch tests on 3 of these demonstrated sensitivity to the tape or its gum.

SPONTANEOUS RUPTURE OF VENOUS ANGIOMA OF THE ORBIT. DR. WILLIAM B. DOHERTY.

In 1910, when the patient was 17 years of age, she stated that while she was working suddenly something "burst" in her right eye. There was intense swelling; the lids were very ecchymotic; the conjunctiva ballooned up with large subconjunctival hemorrhages, and the eyeball protruded. The patient was taken to the New York Eye and Ear Infirmary, and a Krönlein operation was performed by Dr. Emil Gruening. The orbit was found to be full of clots of blood, and a diagnosis of "idiopathic hematoma of the orbit" was made.

During March 1923, about thirteen years later, a similar condition occurred, and the patient was admitted to the Bellevue Hospital in the service of Dr. Charles H. May. At that time the picture was almost identical to that seen in 1910, the patient showing marked ecchymosis of the lids, intense chemosis of the conjunctiva, and exophthalmos of at least 4 mm. Haziness of the cornea reduced the vision to counting fingers at 10 feet (3.3 meters). After recovery there were limitation of motion of the eyeball in all directions, partial symblepharon in the lower lid and marked evidences of a venous angioma. The angioma manifested itself in the lower lid, simulating a bag of blue earthworms which seemed to shine through, and the entire mass could be palpated by the fingers. The patient had a series of treatments with the roentgen rays and radium, given by Dr. G. Allen Robinson, and there has been a gradual diminution of the evidences of the tumor. Vision of the right eye was 20/50 with a -1.00 D. sph. \ominus -1.00 D. cyl., ax. 90, and that of the left eye was 20/20 with a -1.00 D. sph. \ominus -1.00 D. cyl., ax. 75. The fundus showed an area of choroidal atrophy; a roentgenogram of the orbit did not reveal any bony erosion, and the Wassermann reaction was negative. The fields presented a concentric contraction of about 15 degrees. The left eye, with the exception of the error of refraction, was normal.

SCLEROMALACIA PERFORANS. DR. SAMUEL OAST.

This paper was published in full in the April 1937 issue of the ARCHIVES, page 698.

DISCUSSION

DR. ALGERNON B. REESE: While inflammation of the sclera usually produces fibrosis which may lead to great thickening of the coat, rarely, as apparently Dr. Oast's case illustrates, can it lead to a nonreparative process and thereby leave a rarefied area. One ordinarily thinks of scleritis as affecting primarily the anterior part of the globe, because only this portion can be viewed. Inability to recognize inflammation in the posterior portion of the sclera was brought home to me forcibly some time ago. A patient had severe pain in one eye, for which no cause could be found. Finally a rise in the intra-ocular pressure was detected, and the feeling was that perhaps unrecognized glaucoma had been the cause of this pain, so a trephine operation was done. The intra-ocular pressure was controlled, but the pain continued to be as severe

as before. Finally the eye was enucleated because of the pain. Microscopic examination showed inflammation of the posterior portion of the sclera, with thickening of the sclera from three to four times that normally seen.

DR. LEWIS WEBB CRIGLER: About fifteen years ago I had occasion to observe an elderly woman suffering from arthritis deformans. During the course of the disease low grade scleritis developed that involved the entire circumference of the eyeball adjacent to the limbus. The tissues seemed to undergo calcareous degeneration resulting in complete destruction of the entire thickness of the sclera, save a few fine trabeculae which seemed to hold the cornea and sclera together. The tension never rose sufficiently to cause protrusion of the unsupported choroid and ciliary body, which were plainly visible.

The patient died before it was deemed necessary or expedient to remove the eye; thus a rare and valuable specimen was lost. This case appears to come under the classification described by van der Hoeve, in which articular rheumatism was a complicating factor. The characteristic difference in the pathologic features in the two cases just described appears to be that in the one a degenerative process was present, and in the other, a postinflammatory one.

GLAUCOMA AND SYMPATHETIC OPHTHALMIA. DR. BERNARD SAMUELS.

This paper will be published in full in a later issue of the ARCHIVES.

PATHOLOGIC CONDITIONS OF THE ORBIT—ROENTGEN STUDIES. DR. IRVING SCHWARTZ.

Roentgenography of the orbit consists, for the most part, in a careful study of shadows of the bony structures. Slight deviations in position or density may be of decisive diagnostic significance. In some cases roentgen studies may be the only means of deciding whether a disease process is within the orbit or in the periorbital structures, whether it is inflammatory or neoplastic, and, if it is the latter, whether the neoplasm is malignant or benign.

COLLEGE OF PHYSICIANS OF PHILADELPHIA,
SECTION ON OPHTHALMOLOGY

Jan. 21, 1937

CHARLES R. HEED, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

EXHIBITION OF A CASE OF SPONTANEOUS REATTACHMENT OF THE RETINA. DR. WILLIAM ZENTMAYER.

This patient is being presented in connection with Dr. Buchanan's case because of complete spontaneous replacement of the retina following a detachment that was probably exudative in origin.

Vision of the right eye became distorted in April 1930. The first examination was made in September of that year, when it was found that vision equaled 6/15 and the upper part of the field of vision was lost. There was separation of the retina, which began at about the horizontal meridian of the fundus, and extended forward to the ora serrata. There was already marked retinitis striata, a white band demarcating the limit of the separation. The anterior part of the retina was thrown into small folds, and on the temporal side, well forward, there was an area of choroiditis about which there were a half dozen or more gray, almost globular bodies, each about three times the diameter of the central artery of the retina, similar in appearance to the lesions seen in sympathetic ophthalmitis. There were a few striae in the posterior cortex.

By the latter part of October the bodies had disappeared. It was not until August 1932 that it was noted that the retina was entirely back in place and the field was again full. The picture is now one of sequelae of the retinochoroiditis, with the unusual development of retinitis striata. Vision is 6/9.

A CASE OF DETACHMENT OF THE RETINA WITH SPONTANEOUS REATTACHMENT. DR. MARY BUCHANAN.

Miss C. B. R. came to the ophthalmic clinic of the Woman's Hospital in March 1920, five weeks after her discharge from the Mount Sinai Hospital when convalescent from an attack of influenza. She was practically blind, since her right eye showed a detached retina, and her left, a cataract. After six weeks' treatment with a vaccine prepared from mixed influenza bacilli her vision improved to 5/30 in the right eye and 5/60 in the left. On August 9 the cataract was extracted from the left eye, and after this vision with a —2.00 sph. C —2.50 cyl., ax. 85 registered 5/15. In 1925 a cataract was extracted from the right eye, but in 1926, after the extraction of several teeth without the use of gas, the right eye showed an intra-ocular hemorrhage, and it had to be enucleated. In the meantime vision of the left eye became rapidly reduced. In the spring of 1936 the patient reported that there was something hanging over the central sight, and vision was gone. The ophthalmoscope showed a white wall, but no details of the fundus could be seen. Shortly after trauma to her left eye in August she found that she could see better toward the temporal side. Her field, which was taken on October 21, showed a 60 degree boundary below the horizontal line; the field extended 15 degrees above and 20 degrees to the nasal side. Vision was 1.5/60. On Jan. 18, 1937, a large staphyloma and two large patches of sclera could be seen at the nasal side.

A CASE OF UVEOPAROTITIS IN A THIRTY-TWO YEAR OLD NEGRO. DR. ALEXANDER G. FEWELL and DR. LUTHER KAUFFMAN.

A 32 year old Negro had uveoparotitis of about eight weeks' duration. The following features were present: markedly enlarged and indurated parotid glands on each side, facial paralysis on the right and some of the features of uveitis in each eye. Roentgen examination of the chest revealed thickening of the hilus shadows. The blood count revealed leukopenia and eosinophilia.

ILLUMINATION. DR. ALFRED COWAN.

Ophthalmologists should be familiar with the problem of artificial lighting and in agreement with illumination engineers in their endeavor to bring about higher intensities and proper arrangements of lighting fixtures.

DISCUSSION

DR. WILLIAM ZENTMAYER: Since Dr. Cowan has told of occupational illumination and illumination in the home, I wonder whether he would give information as to the illumination of test charts.

DR. ALFRED COWAN: I think that test cards should be illuminated by about 100 foot-candles, with adequately lighted surroundings. Wide variations in the intensities around this range will cause relatively small changes in the visual acuity. Also, with this amount of illumination there will be less fatigue of the patient in those instances in which considerable time is required for refraction by the subjective method.

DR. GEORGE S. CRAMPTON: Because of my experiences in connection with the Illuminating Engineering Society, I recommended this contact on the part of ophthalmologists as a pleasant and scientifically profitable one, as both groups are interested in light as a medium through which to gain their end-results. It would seem advisable to go slowly in adopting the high intensities on test charts, as recommended by some, as the fatal element of glare can readily become a menace under these intensities and defeat the purpose of a reposeful refraction.

DR. ALFRED COWAN: Of course there must not be glare, and Dr. Crampton is correct in his statement that glare will cause fatigue; but there should be no glare in the illumination of test cards if the surroundings are properly lighted. It is important that the room in which a highly illuminated test card is used should be diffusely illuminated in the proper proportion in order to eliminate glare.

EXPERIMENTAL AND CLINICAL STUDIES OF CATARACT. DR. I. S. TASSMAN.

Moerner in 1894 described the chemical analysis of the crystalline lens and its reactions. Later investigations have shown the chemical changes which take place in the crystalline lens during the development of senile cataract. Following this, the results of the investigations concerning the changes which take place in the calcium content in the lens and blood serum of patients with cataract, the cholesterol content, the permeability of the capsule of the lens and the oxidation-reduction system of the lens have been described under the following groups:

1. Experimental cataracts, such as those produced by feeding animals either naphthalene, lactose or galactose.
2. Cataracts produced as the result of either light rays or heat.
3. Cataracts of so-called endocrine origin.
4. Vitamin deficiency, particularly vitamin C deficiency, and its relationship to the development of cataract.
5. Immunologic studies, with reference to cataract.

DISCUSSION

DR. C. A. CLAPP: The first type of cataract that Dr. Tassman has mentioned, which I wish to discuss, is the glassblowers' or radiation cataract. Dr. Tassman has mentioned Professor Vogt's work along this line. There has been much controversy as to whether the opacities which develop are the result of heat or of the infra-red rays. I was privileged to see Dr. Vogt give rabbits radiation with the infra-red rays at 9 a. m., and at 2 p. m. on the same day the lenses showed a general clouding. It did not seem that heat was a factor in the production of the opacities because of the fact that since the rays were passed through a water filter only a slight feeling of warmth was experienced when one held the hand in the ray.

I have also had a series of rats on the vitamin G deficiency diet of Langston and Day, but my rats did not show cataract as frequently as theirs did. In regard to those rats which did show lenticular opacities, I felt that in every case one could detect damage in the capsular epithelium. I am inclined to believe that this damage, by altering the nutrition to the fibers of the lens, caused the opacities.

The results of the experiments on the series of rats placed on the diets of lactose and galactose were also disappointing, since all the rats died before opacities developed.

While Dr. Tassman did not mention cataract following the use of dinitrophenol, I believe this type of cataract also is caused by damage either to the capsular epithelium or to the epithelium of the ciliary processes, either one of which may disturb the nutrition of the lens. Dally has stated the belief that so-called "dinitrophenol cataract" is due not to dinitrophenol but to dinitronaphthol, which may occur as an impurity, since no cases of this cataract have been observed in France, but all have originated in the United States. Experimentation on animals seems to be without results, since so far my animals have failed to show any opacities of the lenses.

p_H AND BUFFERS IN THEIR RELATION TO OPHTHALMOLOGY. DR. J. B. FELDMAN.

This paper will be published in full in a future issue of the ARCHIVES.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION
OF OPHTHALMOLOGY

Feb. 12, 1937

MR. W. H. McMULLEN, O.B.E., F.R.C.S., *President*

THE ACTS OF CLOSING AND OPENING THE EYES. SIR ARTHUR HALL,
F.R.C.P.

Oculogyric movements have been said to occur in 20 per cent of the cases of parkinsonism. In these attacks there is usually a temporary upturning of the eyes, which first became known when

epidemic encephalitis appeared. There are at least two other forms of ocular attack: one, a fixation of the gaze, and the other, inability to open the eyes. The extraordinary feature of the latter form is that when the patient wishes to open the eyes, light tactile contact with the lid breaks the spell, and the eye opens. In the third stage of the disease the patient is unable to lift his arm high enough to touch his eyelid, and then when a friend is asked to touch the lid the eye opens. In the oculogyric attack the eyes do not always turn directly up; sometimes they turn up to one side. In none of these attacks is there any marked wrinkling of the skin of the forehead.

There are certain regulations which must be observed by the eyes when a pattern is formed for binocular vision: The two eyes must act together; they must always be in a state of attention, and the pupils must be kept clear of the eyelids. Sir Charles Bell gave, in 1823, a description of what happens to the eyes when they are closed. An investigation of the eyes of 223 persons in sleep showed that the eyes were up in 65 per cent, straight forward in 29.5 per cent, down in 3 per cent and to one side in 2.5 per cent. Among children, the proportion of those in whom the eyes were up was not so great as the proportion among adults, and among a few blind children examined the proportion was still smaller. It is therefore clear that the eyes may be in almost any position during sleep.

The movements of the eye when the lids are closed may be divided into three types, according to the quantitative ascent of the eyeball: (1) very high; (2) less high, the lids being held, and (3) movements in which the eyeball does not turn up at all and may turn down. Among 900 cases, in 58 per cent the movements were of type 1; in 32 per cent, of type 2, and in 10 per cent, of type 3.

CONGENITAL RETINAL FOLD. MR. ADRIAN CADDY.

A school girl aged 13 showed a retinal fold in the right eye, extending from the disk outward toward the periphery of the fundus, in the direction of 8 o'clock. The eye was slightly divergent, and no blood vessels were seen. This condition of congenital retinal fold has been extremely well described by Miss Mann in the December 1936 issue of the *British Journal of Ophthalmology*, and a similar condition was described by Mr. Jameson Evans at a meeting of the Midland Ophthalmological Society last summer.

DISCUSSION

MISS IDA MANN: Unless the drawing shown is faulty, I do not think this case belongs to the group I described. In all the cases described by me there were on top of the fold branches of the retinal vessels which came up from the surface and, in some cases, ran along its sharp edge. I suggest that the condition in the present case is primarily an abnormality of the vitreous, which has become secondarily attached to the surface of the retina.

MR. MALCOLM HEPBURN: I have observed a case of a condition similar to this in appearance, the scar tissue being definitely choroidal inflammatory scar tissue, and the fibrous tissue going backward in the

form of a band. In this case, however, the tissue is not so evidently scar tissue, as there is not so much proliferation of pigment as in my case.

UNUSUAL OPACITIES IN THE CORNEA. MR. E. F. KING.

A married woman aged 53 came to the hospital a few weeks ago for new reading glasses. Routine examination of the cornea revealed peculiar opacities. They were of two kinds: first, feather-like opacities, chiefly in the temporal half of each cornea, just beneath the epithelium, and, second, deeper and more circular opacities, which were beneath the level of the feathery ones. Apart from early senile cataract, the patient was in good health. These findings suggested hereditary nodular corneal degeneration.

DISCUSSION .

MR. T. HARRISON BUTLER: A case almost identical with this in appearance was presented by Dr. Bamford at a meeting in Derby. In the present case all the opacities seemed superficial, and I think that the condition is chronic epithelial thickening.

Book Reviews

Trachoma. By A. F. MacCallan, C.B.E., M.D., F.R.C.S. Price, 21 shillings. Pp. 225, with 20 illustrations, the majority in color. London: Butterworth & Co., Ltd., 1936.

This book replaces in a more extensive form MacCallan's well known monograph "Trachoma and Its Complications in Egypt," published in 1913. The author's wide clinical experience as director of the Egyptian ophthalmic hospitals and his further activity in the campaign against trachoma since his return to London render him peculiarly fitted for the writing of a treatise on this disease.

Perhaps the most distinctive feature of the monograph is the great number of drawings in color which illustrate the chapters on the clinical manifestations and sequelae of trachoma. Unquestionably they add greatly to the value of the book, particularly the value to ophthalmologists in relatively trachoma-free areas in which opportunities for seeing all stages of the disease are limited. The majority of the drawings were obtained by MacCallan from Cuénod and Nataf in Tunis and from Wilson in Cairo.

The second chapter of the book is devoted chiefly to descriptions of the clinical stages of trachoma according to the classification suggested by MacCallan a number of years ago and now employed by most trachomatologists. Two new conceptions are advanced: 1. The author now describes, on the basis of the observations of Michail, a lymphocytic type of trachoma of stage I, characterized by a generalized subepithelial infiltration with lymphocytes, without the formation of follicles. 2. The term "bleb-like excrescences" is introduced to describe the expressible so-called follicles, resembling sago grains, of stage IIa. The author states that these excrescences are not true lymphoid follicles but cysts of meibomian glands due to blockage of ducts by pressure of subepithelial infiltration and cicatricial contraction. Accordingly, the gelatinous materials expressed from the so-called follicles of trachoma of stage IIa are to be considered not follicular contents, as is generally believed, but débris, sebaceous material, and inflammatory cells from these cysts. In the reviewer's opinion this conception needs additional evidence to substantiate it.

In the chapter on differential diagnosis the conditions which bear a resemblance to trachoma are grouped as common, uncommon, rare and tropical. It is interesting that in the first group are placed such conditions as acute conjunctivitis with follicles and chronic conjunctivitis with corneal vascularization resulting from former interstitial keratitis, while among uncommon conditions are listed spring catarrh and inclusion conjunctivitis. On the basis of the reviewer's experience in three sections of the United States, the relative frequency of these conditions in this country would seem to be reversed.

MacCallan emphasizes in this chapter, as well as repeatedly in other parts of the book, the diagnostic value of slit lamp examination of the cornea for the detection of the early signs of trachomatous pannus. He states that pannus is trachoma of the cornea and that its presence

can be detected as soon as the earliest diagnosis of trachoma of the conjunctiva can be made.

MacCallan apparently considers that no real advances have been made in the therapy of trachoma. In his chapter on treatment most of the new therapeutic measures suggested in the past few years are mentioned but are not recommended. Mechanical expression of follicles and daily swabbings with a 10 per cent solution of copper sulfate are considered most valuable. In cases in which there is secondary bacterial infection, treatment with silver nitrate is recommended. The author states that long-continued oral or tonsillar sepsis has a distinctly bad effect on the disease. The surgical treatment of the cicatricial sequelae of trachoma is described in detail. Diagrams have been omitted purposely.

Sixteen pages of the chapter on epidemiology are devoted to a discussion of the etiology. The work of recent investigators is cited in detail. It is to be regretted that in referring to several reports by joint authors MacCallan names only the senior investigators. No conclusion as to the etiology is drawn, but the virus theory seems to be regarded with the most favor.

The final chapter is devoted to the history of trachoma from the earliest Egyptian records to the present time. Considerable space is devoted to the ophthalmic campaign in Egypt from 1903 to date.

PHILLIPS THYGESON.

Transactions of the Ukrainian Leonard Hirshman Memorial State Ophthalmologic Institute. Volume 3. Edited by E. B. Rabkin. Pp. 178. Kharkov, U. S. S. R.: Ukrainian State Medical Publishing Board, 1935.

This volume consists of thirteen papers written by the pupils and co-workers of Prof. Peter P. Prokopenko on the occasion of the fiftieth anniversary of his medical, scientific and pedagogic activities. The papers are in Russian, but each one is followed by an abstract in English. The volume opens with a greeting to Professor Prokopenko from his pupils and is followed by a short biography of Professor Prokopenko, written by I. O. Merkulov.

The first paper, by E. B. Rabkin, is concerned with the polychromatic plates for the diagnosis of color blindness, which are discussed elsewhere, in a book review in the *ARCHIVES*.

I. I. Merkulov and S. U. Minkin report on the relation between the intracranial and the intra-ocular pressure in two patients. They found no correlation between the intracranial and the intra-ocular pressure, even when the intracranial pressure was acutely increased or decreased.

The results of an investigation on the adaptation to darkness in cases of optic neuritis and of choked disks showed that in the former the adaptation is disturbed, while in the latter group it is normal except in cases in which the condition is very advanced (I. I. Merkulov and A. G. Bereshaja).

K. I. Babenko investigated the ocular tension in the healthy eye when one eye (of a rabbit) was traumatized. If the trauma caused pain the tension in the healthy eye was always increased. The introduction of cocaine locally or procaine hydrochloride retrobulbarly

delayed the rise in tension, and the rise was less marked. General narcosis (induced by chloral hydrate) completely inhibited the rise of tension in the normal eye.

N. O. Tchepurin found that an injury to one eye lowered the adaptation to light in the uninjured eye. Removal or evisceration of the injured eye was followed by a return to normal of the adaptation to darkness in the sound eye.

D. M. Natanson, E. V. Torgovitzkaja and E. S. Gottlieb, in an article which is not suitable for detailed report here, report favorably on the use of tuberculin for tuberculous conditions of the eye.

N. I. Medvedjev reports a new operation for trichiasis. He makes an incision along the intermarginal space of the upper eyelid and then transplants a strip of bulbar conjunctiva into this space. This method is not applicable in cases in which the trichiasis is accompanied by curving of the tarsal plate.

H. B. Stepanova, N. S. Asarova and A. E. Goldfeder report on a biologic method for the isolation of *Bacterium granulosis*.

A. E. Goldfeder describes an interesting operation for trachomatous pannus. The conjunctiva is removed at the affected part of the cornea, but three or four conjunctival bridges are left. Then a flap of cartilage taken from the auricle is placed in the peridectomized space under the conjunctival bridges.

There are also papers on the reduction of diseases of the eye in the Ukraine, by E. B. Rabkin and I. N. Miller; on the constitution of the eye and its relation to the general body constitution, by A. I. Dashevsky; on methods of studying fatigue of the eyes, by A. I. Dashevsky and R. B. Zaretskaja, and on elementary education of school children in the hygiene of the eye, by R. B. Zaretskaja.

W. F. DUGGAN.

Physician, Pastor and Patient; Problems in Pastoral Medicine. By George W. Jacoby, M.D., Past President of the American Neurological Association and the New York Neurological Society. Price, \$3.50. Pp. 390, with 20 illustrations, chiefly portraits. New York: Paul B. Hoeber, Inc., 1936.

Jacoby, long a successful practitioner and teacher of neurology, has previously written two books for nonprofessional readers—"Child Training as an Exact Science," treating of the relationship between the physician and the teacher, and "The Unsound Mind and the Law," on the relationship between the physician and the jurist.

In the present volume his purpose is to discuss without bias the relations between medicine and religion—the physician and the clergyman. He believes that the antagonism between the two has been over-emphasized for centuries and that the real conflict has been between the narrow-minded and dogmatic of either profession, whereas the liberal-minded of each now agree on many points and are cooperating to bring about better health—physical, mental and moral.

The first sections of the book, on the physician's calling and on religion and the patient, comprising 180 pages, are devoted to a historical résumé of the development of medicine from its beginning and to an account of the past and present medical beliefs and practices

of Judaism, Christianity and Islam, as well as of the numerous religions of the East.

The following 150 pages on vital problems confronting the physician and the clergyman treat in detail of the following moot subjects: contraception and abortion, birth control, the questionable sanity of the suicide, divorce, the psychology of the criminal, sterilization, sex education, insanity, euthanasia, vivisection and professional secrecy.

The concluding section, of 40 pages, has as its topic "Where Medicine and Religion Join Hands in Everyday Life."

An enormous quantity of information is assembled in this volume, and debatable matters are discussed impartially, so that the reader, having before him the data and the arguments pro and contra, may form his own conclusions on the many points whereon the clergyman and the popular opinion of the past, as expressed in civil law, are woefully at variance with the physician in his present day beliefs.

WARD A. HOLDEN.

Der Augenbefund in seiner diagnostischen und differentialdiagnostischen Bedeutung bei Tabes dorsalis, Lues cerebrospinalis, multipler Sklerose. By Carl Behr. Price, 7.60 marks. Pp. 60, with 26 illustrations. Berlin: S. Karger, 1936.

In this treatise Behr, one of the most competent and successful investigators in the bordering fields of neurology and ophthalmology, deals with the ocular symptoms of some diseases in which the former frequently not only precede the other neurologic signs and symptoms but are decisive for differential diagnosis. First there is discussed the anatomicophysiologic basis of the ocular symptoms of cerebral origin, especially the anatomic structure of the optic nerve and the pathways transmitting the different stimuli to the pupil. The typical Argyll Robertson phenomenon, which occurs solely in tabes and is therefore an unequivocal sign of this disease, presents a combination of absence of reaction to light with miosis, increased convergence reaction and absence (or decrease) of the sensory and psychic dilatation reflex. Absence of the light reflex without the other aforementioned characteristics is met with not only in tabes but in other intracranial diseases. Absence of the light reflex combined with a tonic, i. e., protracted, convergence reaction, occurs in tabes as well as in epidemic encephalitis. This pupillary disorder resembles an incomplete absolute rigidity of the pupil and is found in oculomotor paresis before the fibers innervating the sphincter iridis are completely paralyzed, so that there is no pupillary reaction to light but a tonic, though sometimes ample, convergence reaction. The reviewer has seen the same behavior of the pupil rather frequently in cases of basal oculomotor paralysis, as well as in nuclear ophthalmoplegia interior, after an incomplete recovery. The pupil was dilated; its reaction to light was absent and never did return. The convergence reaction, however, which was imperfect and slow in the beginning, improved gradually until it was finally normal with regard to the amount as well as the speed of movement. Such cases are discussed at length in the reviewer's "Die Lähmungen der Augenmuskeln," in von Graefe and Saemisch's "Handbuch der gesamten Augenheilkunde," 1932. The different manner in which syphilitic and metasyphilitic diseases are produced accounts for the inexpediency or danger of antisiphilitic treat-

ment in metasyphilitic diseases. Stimulated by the good results obtained in cases of progressive paresis with malaria infection, Behr, eight years ago, started experimenting with intracutaneous injections of very small doses of tuberculin or peptone in the treatment of tabetic atrophy of the optic nerve. He got the impression that by such injections the atrophic process can be retarded, if not brought to a standstill. A rapid progression of the tabetic atrophy may be caused by antisyphilitic treatment, particularly in cases in which the atrophy is combined with papilledema. The same warning holds good if dark adaptation is considerably diminished and the visual field for colors is much contracted, while the limits of the field for white may still be fairly normal. The optic atrophy in association with tabes, as a rule, precedes the impairment of vision, in contrast to the course in disseminated sclerosis. The degeneration of the nerve fibers follows the degeneration of the neuroglial tissue, by which the blood supply is gradually reduced.

In syphilis cerebrospinalis, gummatous tumors, alterations of the blood vessels or inflammation (meningitis) may bring about ocular disorders. Retrobulbar neuritis that causes a central scotoma yields to antisyphilitic treatment. Peripheral contraction of the visual field may be combined with either homonymous or bitemporal hemianoptic defects, according to whether the optic tract or the chiasm is affected. The good results of antisyphilitic treatment are sometimes transitory, since the cerebral substance may be injured by contraction of the blood supply due to secondary shrinkage of the granulation tissues. Paresis of the third nerve is frequent, paresis of its interior branches (ophthalmoplegia interior) being one of the earliest signs of syphilis cerebrospinalis. Congenital syphilis may be the cause of dystrophia adiposogenitalis in children.

In disseminated sclerosis the medullary sheaths of the optic nerve have a predilection for injury, while the axis-cylinders frequently remain intact. As a rule, sclerotic zones in the chiasm, as well as in the optic tract, cause no symptoms, while if located in the optic nerve they bring about a central scotoma, owing to the descending degeneration. A sudden attack of complete blindness or pronounced amblyopia, without the eyeground showing any pathologic changes, is one of the characteristics of disseminated sclerosis. After a short time peripheral vision returns, while the central scotoma remains for a longer period, its size gradually diminishing. Finally, normal vision may be restored notwithstanding the fact that the atrophic discoloration of the optic disk becomes more and more evident. Cocaine and epinephrine administered by tamponade of the nose may hasten improvement in vision. Patients with acute retrobulbar neuritis must be kept in bed six weeks, since the circulation of the infectious cerebrospinal fluid is quickened by physical efforts. Transitory and recurrent paresis of either individual muscles or associated movements occur frequently in cases of disseminated sclerosis, particularly in cases of paresis of lateroversion associated with paresis of the sixth nerve of the same side. Jerky nystagmus in association with lateroversion confined to the eye with the paretic abducens nerve points to a supranuclear pontile lesion involving the sixth nucleus.

Behr's booklet presents so many important facts and considerations that a thorough study of it is highly recommendable.

A. BIELSCHOWSKY.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.

President: Dr. P. Baillart, 66, Boulevard Saint-Michel, Paris (6^e).

Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ost-flandern, Belgium.

All correspondence should be addressed to the President, Dr. P. Baillart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.

Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.

Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.

Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.

Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.

Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.

Secretary: T. Harrison Butler, 81, Edmund St., Birmingham, England.

Place: Birmingham and Midland Eye Hospital.

Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.

Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.

Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.

Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Time: June 11, 1937.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. F. Berg, Uppsala, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.

Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Place: Atlantic City. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.

Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

Place: Hot Springs, Va. Time: June 3-5, 1937.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.

Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.

Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
 Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
 Place: Salt Lake City, Utah. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
 Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
 Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
 Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
 Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
 Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIoux VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
 Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
 Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
 Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
 Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. W. Beals, Weber Bldg., DuBois.
 Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
 Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
 Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
 Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
 Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.

Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Building, Atlanta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.

Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.

Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.

Place: Des Moines.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.

Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.

Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. Coulter Chaffin, 124 S. Illinois Ave., Atlantic City.

Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.

Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.

Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.

Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.

Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.

Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.

Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.

Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
EYE, EAR, NOSE AND THROAT

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.

Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.

Secretary-Treasurer: Dr. Meyer H. Riweh, 367 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.

Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.

Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.

Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.

Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.

Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.

Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.

Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.

Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.

Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.

Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.

Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.

Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.

Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.

Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.

Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.

Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.

Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.

Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.

Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.

Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.

Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.

Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.

Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.

Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.

Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.

Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.

Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.

Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.

Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
 Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
 Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
 Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
 Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
 Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
 Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
 Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
 Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
 Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
 Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
 Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
 Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member in alphabetical order.
 Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
 Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
 Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
 Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
 Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
 Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
 Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
 Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
 Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
 Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
 Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St. New York.
 Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
 OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
 Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
 Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner;
 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
 Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
 Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
 Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
 Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
 Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
 Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
 Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
 Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
 Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
 Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
 Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
 Place: Oscar Johnson Institute Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
 Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
 Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
 Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
 Place: Society's Building, 2180 Washington St., San Francisco.
 Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
 Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
 Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
 Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
 Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
 Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
 Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W. Toronto.
 Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
 Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
 Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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